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THE DEVELOPMENT OF ROOT-KNOT NEMATODE GALLS

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INTRODUCTION

Many nematodes penetrate the tissues of growing plants for the purpose of securing food. Some species in the process of feeding move about through the plant, causing considerable destruction of cells, and may appropriately be regarded as "grazers." In such cases abnormal structures frequently result that usually are referred to as galls. These are brought about in part by irregularities of growth resulting from tissue destruction and in part by cellular hypertrophy. In other cases a highly developed interrelationship has been evolved between the plant tissues on the one hand and the nematode on the other, and both host and parasite react in a definite and characteristic manner. The so-called root-knot nematode, *Heterodera marioni*,[1] is an example of this type of highly specialized, obligate parasite.

Over 900 different species of plants, including both monocotyledonous and dicotyledonous, are known to serve as hosts for the root-knot nematode. The parasite usually penetrates such underground parts of the plant as roots, rhizomes, and tubers, where it induces the development of abnormal growths or enlargements. In some instances the basal portion of the stem is invaded. The size and character of these enlargements vary in different plants. In the case of Gerbera and, to a less extent, Cyclamen, external manifestations are sometimes small and inconspicuous or even lacking, while in the case of *Thunbergia laurifolia* and rhubarb, enormous structures nearly 2 feet in diameter may develop. It is interesting to note that the unusually large galls reported by Steiner, Buhrer, and Rhoads (14) developed at the base of the stem.

The adult female parasite usually lies with its head imbedded in the vascular cylinder, the posterior part of the body extending into the cortex. The eggs, after deposition, are surrounded by a gelatinous substance. As they accumulate at the posterior end of the parasite (Fig. 8, C) the cortical tissue is often ruptured and the eggs are pressed out and may occur in masses at the surface of the root. Two kinds of infestation take place.

[1] *Heterodera marioni* (Cornu, 1879) Goodey, 1932; synonym, *Heterodera radicicola* (Greeff, 1872) Müller 1884 of authors. Not *Anguillulina radicicola* (Greeff, 1872) Goodey, 1932; synonym, *Anguillula radicicola* Greeff, 1872.

1. Eggs may hatch within the root and the larvae migrate into adjacent tissue where they become established. This type of infestation is evidently of common occurrence, often resulting in the formation of unusually large galls.

2. Eggs may hatch at or near the surface of the root and the larvae escape into the soil. These larvae migrate to, and enter, new roots. The present paper deals exclusively with galls produced by this type of infestation.

It has been demonstrated repeatedly that at or near the root tip is a favored point of entrance. However, penetration of the larvae evidently is not confined to this region. Galls occur on rhizomes, tubers, and various parts of the plant under circumstances that render unlikely the entry of the parasite at a growing point. Nevertheless, a very large percentage of the galls found on most plants is initiated by larvae that penetrate near the tips of growing roots.

About 60 to 72 hours after the larva has become established in the root tissues, cells lying adjacent to the head of the parasite begin to undergo a change. They increase in size, the nucleus divides and there are finally formed the peculiar, multinucleate structures, long known in the literature as giant cells.

LITERATURE

Müller's excellent paper (9) is the earliest comprehensive study of the morphology and development of *Heterodera marioni.* The structure and development of the galls also are discussed in a general way, those that occur on *Musa rosacea* being selected as examples. This investigator noted the proliferation of cells in the region outside the vascular cylinder and the disarrangement of the vascular elements, but failed to mention the giant cells.

Frank (5), in his study of gall development, secured his material from various cultivated plants including clover, *Trifolium pratense;* beets, *Beta vulgaris;* lettuce, *Lactuca sativa;* pears, *Pyrus communis;* and teasel, *Dipsacus fullonum.* The writer notes that, in general, cells in the region of invasion retain their ability to divide for a considerable time. There is formed around the region of the central cylinder a layer of short, thin-wall, cells, some of which may later be differentiated into xylem elements. With regard to this Frank writes as follows: "Die Folge ist, dass, wenn in dem umliegenden meristematischen Gewebe einzelne Zellen zu getüpfelten Holzelementen werden, dieselben so wie ihre Mutterzellen eine mehr kurze Form besitzen und diese Zellengruppen unregelmässig verschoben liegen, so dass ihre Zellen auf dem Querschnitte in den verschiedensten Richtungen zur Längsausdehnung durchschnitten erscheinen." Frank further notes that the walls of cells adjacent to the cavity occupied by the parasite usually remain thin. No mention is made of giant cells.

Treub (17) briefly describes the effects of *Heterodera marioni,* which he

erroneously named *H. jarvanica,* on the roots of sugar cane. This investigator mentions that the larvae are able to enter roots with but slight injury to the cells. He also notes a tendency for lateral roots to occur at the region of the gall, but believes this results from larvae migrating to regions where lateral roots are forming. Treub describes and figures the giant cells. Commenting on the division of their nuclei he writes, "Jamais je n'ai rencontré jusqu'ici dans les grandes cellules, des noyaux présentant des phases de la division indirecte. Par contre, j'ai vu plusieurs fois – – – –, où les noyaux font l'effet de se multiplier par division directe."

Atkinson (1) studied the life-cycle of *H. marioni* and the effect of its presence on the roots of various plants including cotton, tomato, potato, and parsnip. He reports that the parasites are not confined to any particular tissue element or system, but locate at various points in the root, including the central cylinder, cambium, parenchyma, or even the bark. The mature female cyst often is protected by only a thin layer of dead peripheral tissue or may even be exposed. Atkinson reports that all the tissue elements of the diseased root undergo hypertrophy and some are subject to special changes in form and direction of growth. Parenchyma cells whose tangential diameter is normally greater than their radial, are so changed that their radial diameter is greater, a change that seems to take place in nearly all the parenchyma of the gall, whether near the cyst or distant from it. No mention is made of giant cells nor does Atkinson describe or figure any structures that can be recognized unmistakably as such.

Queva (12) studied the effect of *Heterodera marioni* on the roots of *Dioscorea illustrata.* The parts of the root system selected for study were the nonfleshy portion that connects the fleshy roots with the main root system, and the fleshy roots themselves. In the first instance the parasites locate at the periphery of the primary vascular cylinder. In regions not occupied by galls the vascular cylinder is composed of numerous, radially arranged, primary vascular bundles ("faisceau multipolaire"). In a series of transverse sections, as one approaches the gall, secondary vascular tissue appears. This is formed by a group of cells in the pericycle located on the outer margin of each primary xylem bundle. The innermost cells of each of these groups, those adjacent to the primary xylem bundle, become differentiated into secondary xylem, while the outermost cells become differentiated into phloem. There are thus developed bundles of secondary vascular tissue forming a more or less continuous layer surrounding the vascular cylinder. Near the middle of the gall the vascular tissues of the central cylinder, according to Queva, are no longer recognizable as such, the cell walls not being characteristically thickened. The secondary vascular tissue in this region is separated from the primary vascular cylinder by several layers of newly formed cells and it is from these that the giant cells are derived.

Beille (2) studied the structure of galls on the roots of *Papaya gracilis*. Here the parenchymatous tissue within the gall divides rapidly, forming a mass in which are embedded the giant cells. The cortex sloughs off and the endodermis and pericycle become unrecognizable. The giant cells are limited exteriorly by vascular tissue of new formation, in which the phloem is reduced, but the xylem, composed of short reticulate elements, is more abundant. According to this author, the walls of the giant cells disintegrate and allow the protoplasmic contents of adjacent cells to intermingle. Of special interest is his statement that the body of the parasite, or cyst, is enclosed in 8 or 10 layers of suberized cells.

The galls studied by Molliard (8) were from the roots of "Melon," Coleus and Begonia. This writer notes that, after invasion, the root tip may be arrested in its growth and also that lateral roots frequently develop near the region of invasion. In the melon the giant cells are surrounded by a layer of small, thin-wall cells that, in certain regions, eventually become thick-wall xylem elements. The number of such regions corresponds to the number of primary vascular bundles in the normal root. Molliard notes the irregularities in the form and number of giant-cell nuclei and their subsequent coalescence and disintegration. He also discusses the function of giant cells in supplying food to the parasite.

Tischler (16), studying galls on the roots of *Circaea lutetiana,* devoted his attention primarily to the cytological aspects of giant-cell formation, particularly the methods of nuclear division. He concluded that during early stages of development the division of giant-cell nuclei is by normal mitosis. After a certain period, however, there follows division by amitosis and by fragmentation.

For information regarding the formation and structure of the giant cells we are especially indebted to Němec (10). According to this investigator, inward migration is intercellular, the larvae never boring through cells, a fact that accounts for the absence of dead cells in the region of their path. As long as a larva remains in the periblem no further changes take place in the root cells. As soon as the head is inserted into the plerome the cells close to the mouth opening of the larva immediately commence to enlarge, their plasma content increases and their nuclei divide, without, however, the formation of separating walls. There are formed, therefore, early in the development of the gall, multinucleate giant cells. Only cells of the plerome undergo this transformation.

Němec is inclined to attribute the formation of giant cells to the stimulating action of some substance secreted through the mouth of the parasite. He considers the possibility of its being brought about through the withdrawal of substances by the nematode but regards this as unlikely as other parasites withdraw substances from plant cells, as for example fungi, without stimulating the formation of giant cells.

Němec finds that the division of giant-cell nuclei takes place by mitosis. Following nuclear division, but early in the life of a giant cell, nuclei may coalesce. Various stages of this phenomenon were observed. In some cases nuclei were fusing in pairs or, in the case of older cells, all the nuclei might be massed together near the center of the cell. Thus the older the cell the fewer nuclei it is likely to contain. Němec regarded it as probable that cases interpreted by Tischler as nuclear division by amitosis or by fragmentation were, in reality, stages of nuclear coalescence.

Kostoff and Kendall (6), working with galls on the roots of Nicotiana hybrids, find that in the early stages of invasion multinucleate cells may be formed by nuclear division without accompanying cell division. However, they believe that a coalescing of cells following dissolution of cell walls may be more generally responsible for the formation of giant cells. This process becomes more and more evident with the continuation of parasitism. Cell walls near the oral region of the parasite become increasingly thinner until finally the content of the cells is removed.

These investigators contend that a secretion produced by the salivary (esophageal) glands of the parasite increases permeability in plant tissues, which results in an exosmosis and brings about an accumulation of food in the region of invasion. "As a consequence of the presence of this accumulation of nutrition the growth of the plant tissues is accelerated in these regions and is morphologically expressed by the swelling, or gall, on the root. At the same time however, the other organs of the plant are deprived of nutrition and their growth is inhibited. The increased permeability is instrumental in bringing about a localization of the foreign substances but the plant suffers a loss of nutrition in the substances so carried from the surrounding tissues to the area of invasion, while the parasite receives a great store of nutritive material in a very small region about its head, as is shown in the rich granular cytoplasmic contents in these regions."

The *Chromatinballung* of Tischler and the *Kernverschmelzung* of Němec, according to these authors, is brought about by agglutination accompanied by proteolytic processes. Conformations referred to by Tischler and others, as due to *Amitotische Zweiteilung, Knospung, Sprossung* or *Fragmentation,* are probably early stages of nuclear fusion. This is in agreement with the opinion of Němec.

Cook, in a recent paper (4), discusses the development of root-knot nematode galls on tomato, tobacco, and coleus. He compares his interpretations with those of Atkinson and, like the latter, makes no mention of giant cells.

Saran (13) recently has published a brief account of deformities on the roots of *Hibiscus esculentus* in India caused by *Heterodera marioni.* The tendency for lateral roots to be formed at the region of invasion is noted, but giant cells are not mentioned.

METHODS

The following observations were made on roots of the tomato that were infested in the laboratory. Seed was germinated in small flower pots containing sand. As soon as the cotyledons emerged and when the radicles were 2 to 4 cm., long the pot was submerged in a pan of water and, by manipulating, the seedlings were floated free from the sand. Each seedling was laid on the bottom of a Petri dish with the tip of the radicle in a small drop of water. Into this drop were placed newly hatched larvae of the nematode. The entire radicle was then covered with fine dry sand, which was moistened afterwards with water. The Petri dishes were then covered and kept at room temperature for 24 hours and then each dish was filled with water and the seedlings floated free from the sand. In order to wash off any parasites that had not yet penetrated, but that might still be adhering to the surface of the root, the seedlings were rinsed in a second dish of water. Except for cases where the root was killed and fixed immediately after being exposed to larvae for 24 hours, the seedlings were planted in flower pots and transferred to the greenhouse. Every precaution was taken to avoid injuring the radicle during manipulation of the seedling.

The exact age of each gall, therefore, was known to within the limits of 24 hours. Where the root was to be killed and fixed within 24 to 48 hours, several larvae were used, usually about 10. In all other cases 2 larvae were used.

ENTRANCE OF THE LARVAE

In every case penetration took place close to the root tip, but, inasmuch as the larvae were placed at this point and to have entered elsewhere would have necessitated migration along the root, no particular significance can be attributed to this fact. Entrance through the root cap and migration to a position back of the growing point occurred frequently.

There is unquestionably a tendency for the larvae to push the cells apart and pass between them (Fig. 1, A). It is overstating the case, however, to say that cells are never destroyed. Instances have been observed, especially in the apical meristem or the region immediately behind it, where cells are clearly being crushed and destroyed (Fig. 1, B). Cases also have been noted where the parasites appear to have passed through cells instead of between them. These occurred in the central cylinder after some elongation of the cells had taken place. The larva, in migrating along the root, had apparently broken down the end walls and passed through a row of these cells. Nevertheless, migration of the larvae is largely intercellular, as Němec states, and instances to the contrary should possibly be regarded as exceptions, although probably of not infrequent occurrence. Certainly, the destruction of cells, when compared with that caused by the passage of many other nematodes, is surprisingly slight.

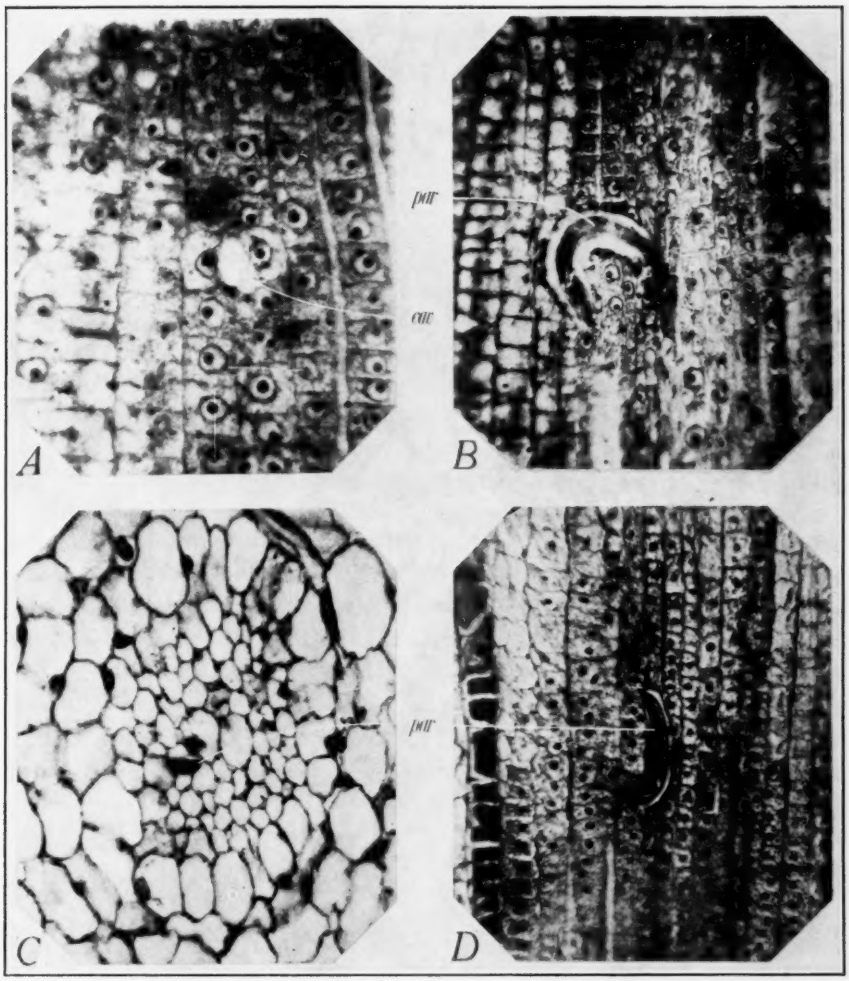

FIG. 1. A. Longitudinal section showing opening (*cav*) caused by migration of larval parasite through root. ×400. B. Longitudinal section showing cell destruction by the entering larva (*par*). ×280. C. Transverse section showing head of larva (*par*) deep in central cylinder. Nearby cells show no noticeable abnormal change. ×400. D. Longitudinal section showing head of parasite (*par*) in plerome at about the beginning of region of elongation. Nearby cells show no noticeable abnormal change. ×280. All sections made after roots had been exposed to infestation for 24 hours.

About all that can be said regarding the final position assumed by the parasite is that its anterior end usually, but not always, lies between the cells of the plerome. Often the posterior end of the body extends into the periblem and, as the root develops, into the cortex. This, however, is not always true, for the entire body of the parasite frequently lies in the plerome parallel with the rows of cells that constitute this region.

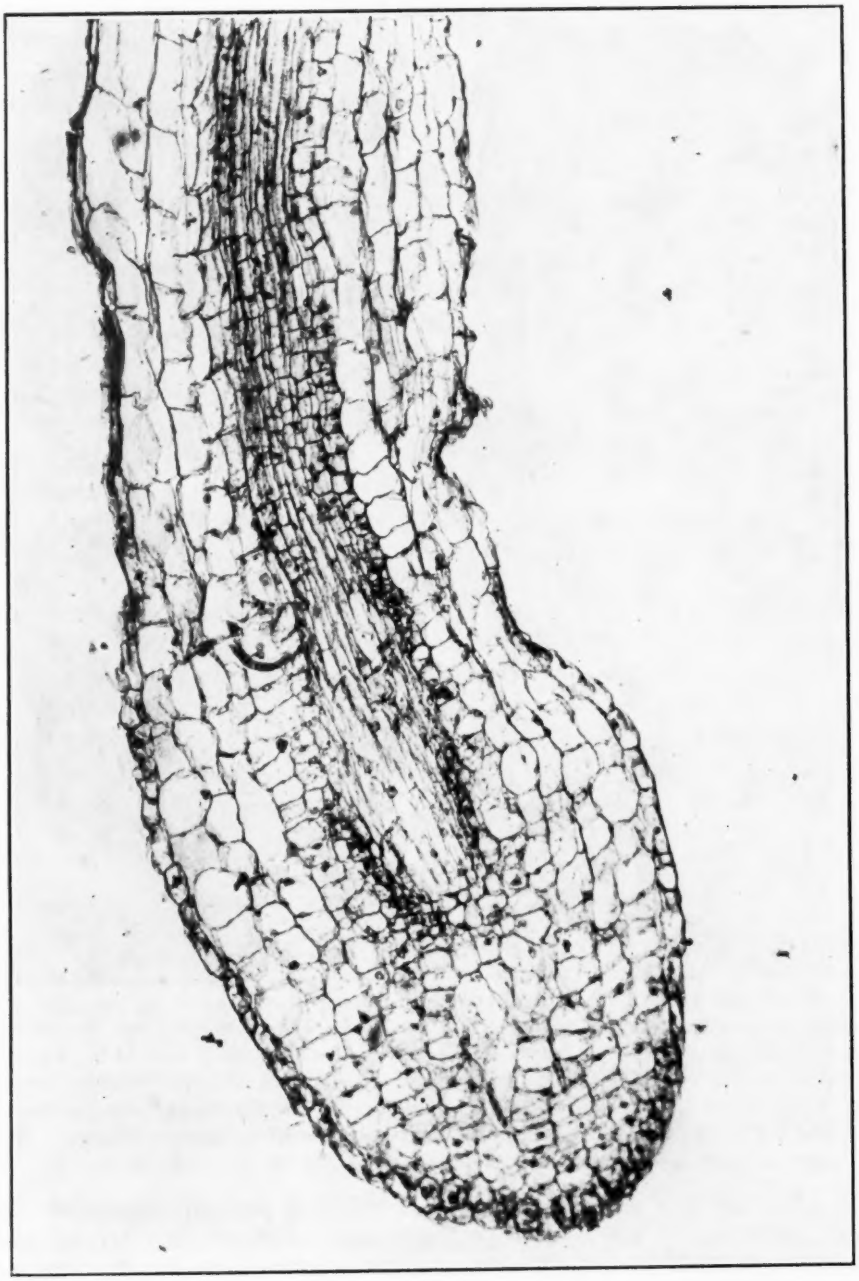

FIG. 2. Longitudinal section through root exposed to infection for 24 hours during which time several parasites entered. The abnormal appearance near the tip is not due to the section's having been cut tangentially or obliquely, as one might at first suspect. ×138.

IMMEDIATE REACTION OF THE PLANT TISSUES

The reaction of the root tissues to the presence of the parasite often is very prompt, although its promptness and extent depend, to some degree, on the number of parasites that enter. The first noticeable change to take place is hypertrophy of the cortical cells. This is not confined to cells lying adjacent to the path of the larva, but those some distance away also may be affected. When several larvae have entered simultaneously, considerable areas in the cortex may show, pronounced cellular hypertrophy at the end of 24 hours. Cells of the pericycle and endodermis, when lying near the path of the larva, will sometimes show slight hypertrophy (Fig. 3, A).

While in many instances a root continues to grow in a more or less normal manner after infestation, sometimes growth is retarded or even stopped, a fact noted by Molliard (8). It is doubtful if the penetration of one larva will ordinarily affect growth to any extent, but when a considerable number enter simultaneously growth may be stopped within 24 hours. The resulting gall will be located, pendulum-like, at the end of the root. Such galls occur frequently in nature.

One root, killed and fixed after being exposed to infestation for 24 hours and harboring several larvae, showed noticeable abnormalities (Fig. 2). Cap cells, for the most part, were lacking. A group of cells near the tip possessed a moderately dense protoplasm, but were vacuolated to some extent and appeared not to be dividing. Cells immediately back of these showed hypertrophy and also were considerably vacuolated. A partly differentiated central cylinder made up of somewhat elongated cells extended far down toward the root tip, where it ended rather abruptly. The surrounding parenchyma cells of the cortex showed hypertrophy to some extent. It would appear, in such cases, that the presence of the parasite suppresses mitotic activity in the apical meristem and hence retards or terminates growth.

One of the first things noticed regarding the galls experimentally produced for this study was the frequency with which lateral roots occurred at the point of infestation. Unfortunately, exact counts were not made, but in fully 60 per cent of the plants from 1 to 4 lateral roots developed in the region of the gall. They were far too common to be explained on the basis of coincidence. This fact has been noted by Treub (16), Molliard (8), Saran (13), and others. Twenty-four hours after infestation one frequently finds, in the region of the parasite, cells of the pericycle undergoing division. In this instance the presence of the nematode seems to stimulate mitotic activity in the pericycle. Once cell division is initiated it proceeds in a more or less normal manner and lateral roots may be formed.

ORIGIN OF THE GIANT CELLS

In the origin of giant cells the first cells affected usually are those of the plerome lying adjacent to the head of the parasite. Larvae frequently be-

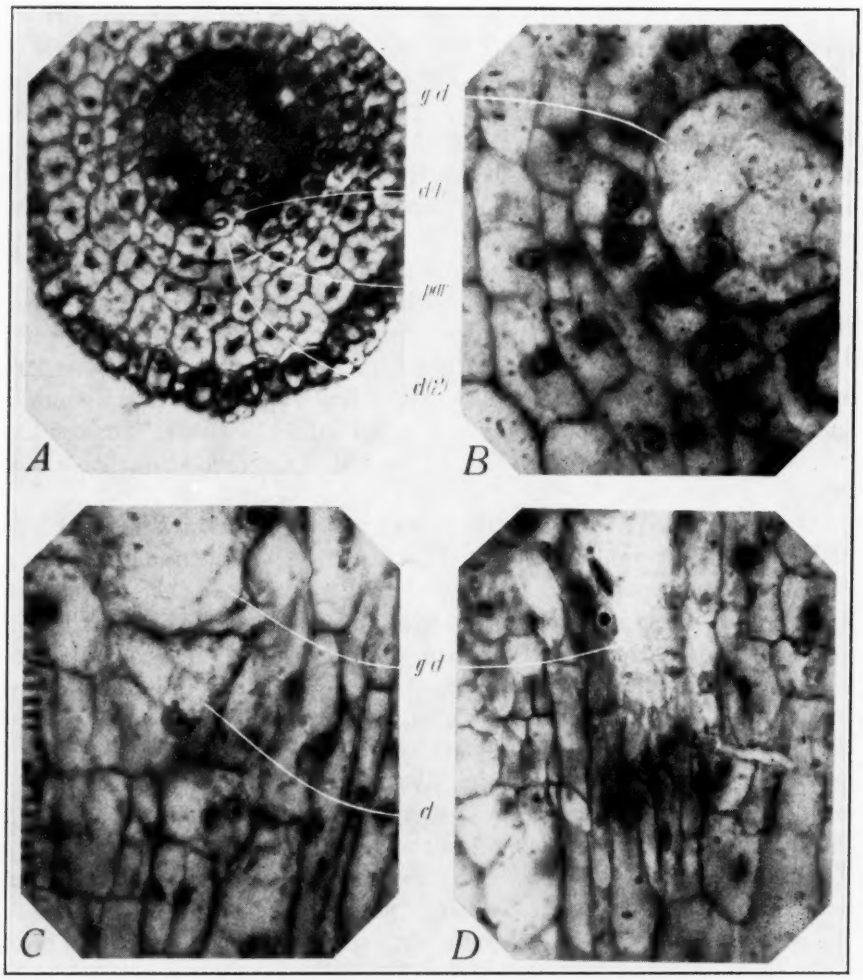

FIG. 3. A. Transverse section through root exposed to infestation for 24 hours. Body of parasite (*par*) is cut transversely where it lies between endodermis and pericycle. In both layers adjacent cells are noticeably enlarged (*cl*(*1*) and *cl*(*2*)). Note intercellular position of parasite. ×280. B. Transverse section through 4- to 5-day gall. Lying adjacent to the giant cell (*g cl*) are cells whose nuclei already have assumed the characteristics of giant-cell nuclei. ×556. C. Longitudinal section through 4- to 5-day gall. Cells (*cl*) adjacent to giant cell (*g cl*) are undergoing change, the walls still are fairly distinct. ×400. D. Longitudinal section through 4- to 5-day gall. Cell walls in region adjacent to giant cell (*g cl*) are disappearing. ×400.

come established only a short distance behind the apical meristem. Not all take up this position, as some either enter or migrate farther back. Nevertheless, at the end of 24 hours, larvae frequently are found in the plerome, between the growing point and the region of elongation (Fig. 1, D); and it seems evident, in such cases, that the stimulus that ultimately results in giant-cell formation becomes operative before the cells of the plerome have proceeded far in their normal differentiation.

As the root grows and the cells of the central cylinder elongate some of the cells near the head of the parasite undergo little change. In many instances these are cells that, in normal development, would contribute to the formation of vessels. Such cells elongate but slightly, or not at all, and their walls do not become thickened. Thus, for the first 48 to 60 hours, the stimu-

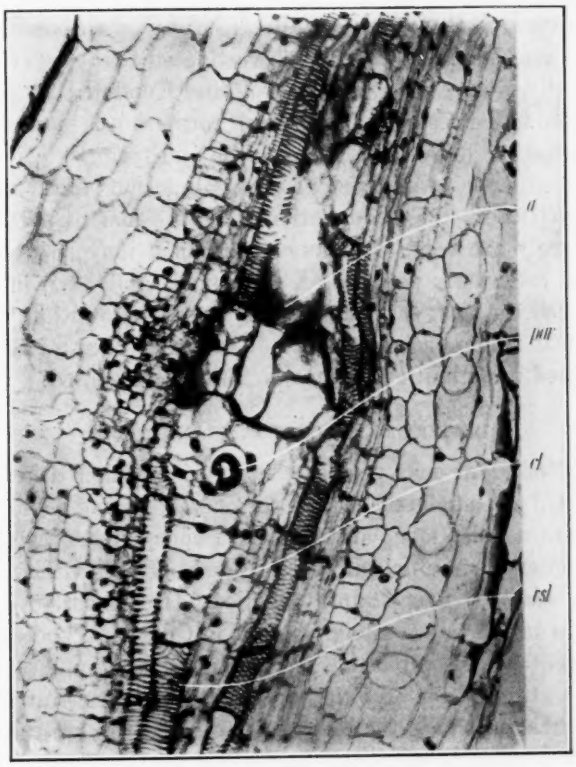

FIG. 4. Longitudinal section through 8- to 9-day gall. One of the vessels (*vsl*) ends abruptly at a cross wall and the 3 succeeding cells, which would normally have formed its continuation, have remained undifferentiated. One of these cells (*cl*) has divided parallel with the main axis of the root and a delicate separating wall has formed. The body of the parasite is cut transversely (*par*) and the head lies near the point marked *a*, where a giant cell is forming. ×150.

lative effect of the parasite on these cells is to *retard differentiation.* At the end of this period some of these undifferentiated cells begin to enlarge and division of the nucleus may take place. The first division may be parallel to the main axis of the root and a delicate cell wall sometimes is formed (Fig. 4), which, however, soon disappears. The original cell walls also gradually disappear and the protoplasmic contents of adjoining cells coalesce to form the beginning of a giant cell. In the tomato, therefore, giant cells are usually initiated in the vascular cylinder and frequently derived from cells that normally would have taken part in the formation of vessels (Figs. 4, 5, 6, A). However they may occasionally be formed in the cortex (Fig. 6, C) but in such cases the writer has not observed any tendency for them to grow inward and thrust aside the vascular elements as is said to take place in galls formed by *Heterodera schachtii.*

No attempt was made to study the details of nuclear division. This phase of the subject has been more carefully investigated than any other, especially by Tischler (16), Němec (10), and Kostoff and Kendall (6). All agree that nuclear division in early stages of giant-cell formation is by mitosis and there seems little reason to doubt that the amitotic and fragmentation divisions of Tischler were either mitotic divisions of an abnormal character or nuclei in early stages of fusion. Němec mentions the fact that nuclei in the process of division rarely are seen. In this connection it may be pointed out that coalescence of cells takes place at an early stage and, even in a 6- to 7-day gall, a giant cell, in most cases at least, is composed of what was originally several cells. It seems probable that this fact may account for more of the nuclei contained in a giant cell than was hitherto supposed.

DEVELOPMENT OF THE GIANT CELLS

The nuclei of giant cells possess characteristics that differentiate them, to some extent, from nuclei of cells in the corresponding region of a normal root. They are usually large and contain a conspicuous, deep-staining nucleolus. Frequently, 2 nucleoli are present and sometimes, in addition, several other smaller, deep-staining bodies. Occasionally, a nucleolus is lacking, but in its place are many, smaller but distinct, bodies. These conditions apparently are comparable to those described by Tischler (16). In a transverse section of a 4- to 5-day gall one of the first things noted is the prominence of the nuclei in the region occupied by the head of the parasite. This not only applies to giant-cell nuclei but also to those in surrounding cells.

After the coalescing of the first few cells the walls of other cells near the periphery of a giant cell become indistinct. In some places the giant cell has a somewhat definite contour, but at other places there is no distinct line of demarcation between it and adjoining tissue. Adjacent cell walls gradu-

ally break down and the protoplasmic contents of the cells, together with the nuclei, unite with the giant cell (Fig. 3, C & D). Often these nuclei have already assumed the characteristics of giant-cell nuclei (Fig. 3, B). When an area composed of small cells is absorbed a group of nuclei is added that seems out of proportion to the size of the area invaded.

At the beginning of giant-cell formation it is usually several adjacent members of a row of undifferentiated cells in the central cylinder that first coalesce through the dissolution of the separating cross walls. This same tendency persists throughout the early stages of development with the result that giant cells tend to extend longitudinally along the central cylinder rather than laterally into the parenchyma. Nevertheless, the surrounding parenchyma is invaded to some extent; in fact, as noted later, giant cells may be formed exclusively from this tissue (Fig. 6, C).

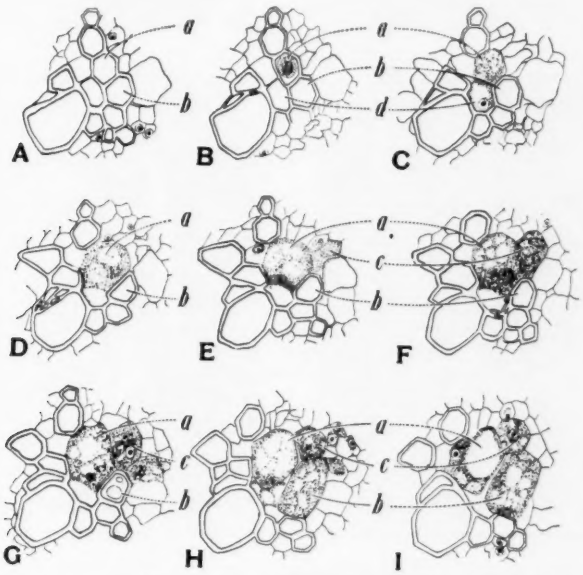

FIG. 5. From a consecutive series of transverse sections, 10 μ thick, through 4- to 5-day gall. A. Section farthest from the parasite. Xylem elements *a* and *b* appear normal with thickened walls. B. Xylem element *a* contains a dense protoplasmic mass but the walls are thickened. Xylem elements *b* and *d* appear normal. C. The beginning of a giant cell shows in the place of xylem elements *a* and *d* without a wall to separate the two. Xylem element *b* remains normal in appearance. D. About the same condition as in C; xylem element *b* is normal in appearance. E. A third giant cell (*c*) makes its appearance, apparently having been derived from an adjacent, thin-wall cell. F. Giant cells *a* and *c* are separated by a fairly distinct wall; xylem element *b* remains unchanged. G. This section includes a cross wall in xylem element *b*. H. Xylem element *b* is now replaced by by a giant cell derived from cells that normally would have formed a continuation of this element. I. Three fairly distinct giant cells are seen. The head of the parasite has not yet been reached. ×230.

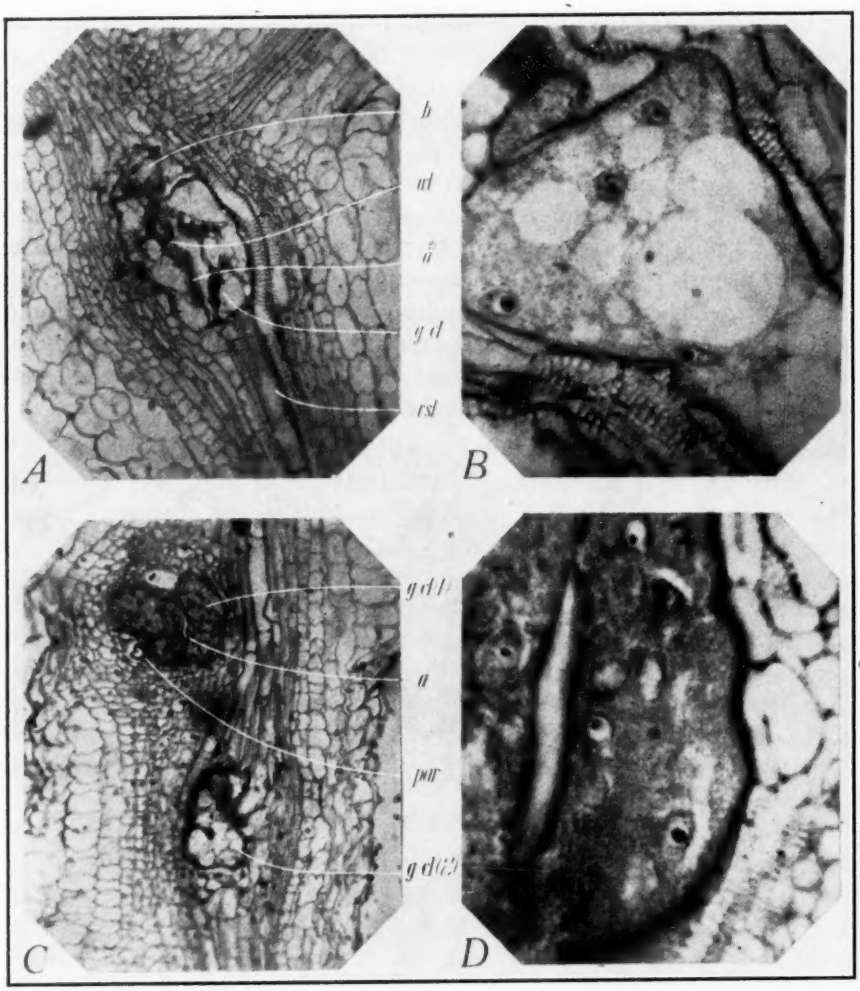

FIG. 6. A. Longitudinal section through 8- to 9-day gall. One vessel (*vsl*) ends abruptly at a cross wall and the cells that normally would have formed its continuation are transformed into a giant cell (*g cl*). Note remnant of cross wall (*wl*). Two more or less distinct giant cells are coalescing at *a*. At *b*, a condition exists similar to that shown in Fig. 3, C, D. A lateral root has developed. ×100. B. Longitudinal section through 10- to 11-day gall, showing typical giant cell. Nuclear membranes are beginning to disintegrate. ×280. C. Longitudinal section through 8- to 9-day gall. Note large number of nuclei in giant cell (*g cl (1)*) formed outside vascular cylinder from small-cell parenchyma as compared with giant cell (*g cl(2)*) formed in vascular cylinder. The parasite (*par*) is cut transversely and the head lies at *a*. ×100. D. Giant cell in a 30- to 31-day gall. Many nuclei have disintegrated. ×280.

Even giant cells of the same age vary considerably in appearance, depending on the character of the cells from which they were derived. Usually they contain, when first formed, large clear areas separated by strands of protoplasm (Fig. 4). As time goes on the protoplasmic network usually becomes denser and the spaces smaller until, at the end of 30 to 40 days, they are more nearly homogeneous in appearance (Fig. 6, D). There is a noticeable tendency, in all stages, for that portion lying close to the head of the parasite to have a dense protoplasm and to stain deeply.

During the first 10 to 20 days the giant cell continues to invade adjacent tissue, coalescing with cell contents after the dissolution of cell walls. As already noted, this does not take place equally in all directions, some areas evidently being more susceptible than others. As the giant cell becomes older this invasion of tissue gradually lessens until, after about 40 days, it appears to have largely stopped.

There are formed in each gall several, usually from 3 to 6, giant cells that remain as more or less distinct units (Fig. 7, D). Giant cells coalesce with the contents of adjacent cells, and, to some extent, with one another (Fig. 6, A), but the writer has never noted an instance, in galls up to 40 days old, where all the giant cells had united to form a single unit, even though they usually lie adjacent to one another.

When examining infested roots at the end of 24 hours it is, of course, impossible to determine whether the position in which the larvae are found is the one in which they would have remained or whether further migration would have taken place. While most of the larvae have the head deep in the central cylinder (Fig. 1, C, D), one occasionally is found with its head in the cortex or between the pericycle and endodermis. That larvae, in some instances, remain in the cortical region, is indicated by the fact that giant cells sometimes are found outside the vascular cylinder (Fig. 6, C).

It has already been suggested that giant cells possess numerous nuclei due largely to the fact that numerous cells contribute to their formation. If this be true it would be expected that where these cells are small and consequently numerous the giant cells would contain a larger number of nuceli than where the cells were large and fewer in number. It is interesting to note, therefore, in the case of a giant cell formed outside the vascular cylinder (Fig. 6, C) from small parenchymatous cells, that the nuclei are very numerous.

REACTION OF ROOT TISSUES SURROUNDING THE GIANT CELLS

Initial stages in giant cell formation take place at about the time when protoxylem is being formed. Outside the central cylinder, and eventually surrounding it, there is a layer of small-cell parenchyma. This, for the most part, originates from the pericycle and outgrowths from it form the

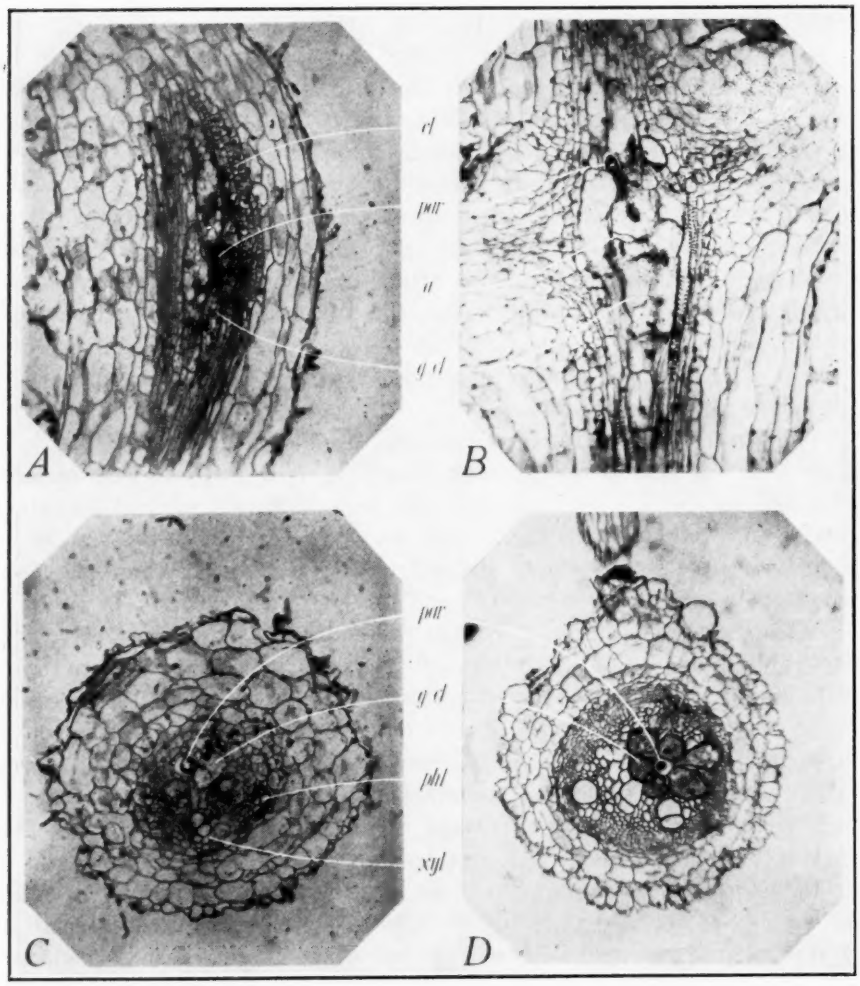

FIG. 7. A. Longitudinal section through 6- to 8-day gall. Note layer composed of small cells (*cl*) bordering the giant cells (*g cl*). ×100. B. Longitudinal section through 7- to 8-day gall. The parasite (*par*) is cut transversely and the head lies near point marked *a*. Two lateral roots have developed. ×100. C. Transverse section through 4- to 5-day gall near head of parasite (*par*). Phloem (*phl*) has formed on both sides in about normal position but xylem (*xyl*) on only one side. On the side of the root where the body of the parasite (*par*) extends into the cortex no xylem has formed. ×100. D. Transverse section through 30- to 31-day gall. Note giant cells (*g cl*) grouped around head of parasite (*par*). ×50.

lateral roots that so frequently develop in the region of infestation. Some of the tracheids or vessels are interrupted in the region of the gall, a fact already noted. Others pass around the giant cells, somewhat pushed out of position, but with their continuity more or less undisturbed. Such continuous vessels, however, usually are few in number. A transverse section through the middle of a 6- to 8-day gall will sometimes show differentiated xylem at both xylem points, frequently at only one and occasionally none at all. Xylem usually fails to differentiate on the side of the root where the body of the parasite extends into the cortex (Fig. 7, C). As the gall becomes older, additional xylem elements are formed. They usually are derived from cells of the surrounding parenchyma. (Fig. 8, B). As these cells are small and irregular in shape, the resulting xylem, as noted by Frank (5), is composed of short, irregular, reticulate elements without direction or recognizable arrangement. It is to this that Beille (2) also refers when he says that the giant cells are limited externally by vascular tissues of new formation in which the xylem is composed of short, reticulate elements.

Usually situated in the vascular tissue, more or less surrounded by xylem, the giant cells eventually come into contact with the thickened walls of these elements. The question arises as to what effect, if any, the giant cell has on these thickened walls. That they are affected, at least to some extent, is indicated by the fact that portions of the walls lying adjacent to giant cells frequently stain differently from the walls elsewhere. Instances were observed also where certain walls of the xylem elements near a giant cell become greatly thickened or swollen. These swollen walls stain a deep red with Flemming's triple stain in contrast to the purplish to blue color usually assumed by such elements. In some cases these modified cell walls blend into the giant cell, as if the substance of the wall were being absorbed (Fig. 8, A). The appearance of such conditions, when seen in stained sections, leads one to suspect that this change is brought about by a substance, produced by the parasite, that seeps between the cells, entering in some places but not in others.

If proteolytic processes result from substances secreted by the nematode, as Kostoff and Kendall (6) suggest, these substances would, when first secreted, occur between the cells. The head of the parasite lies between the cells and there is no evidence that the stylet pierces adjacent walls. Hence cell-wall dissolution may be brought about in this manner, rather than through any direct influence of the giant cell itself.

GENERAL CONSIDERATIONS

One of the first effects of *Heterodera marioni* on root development in the tomato is the suppression, in some instances, of mitotic activity in the apical

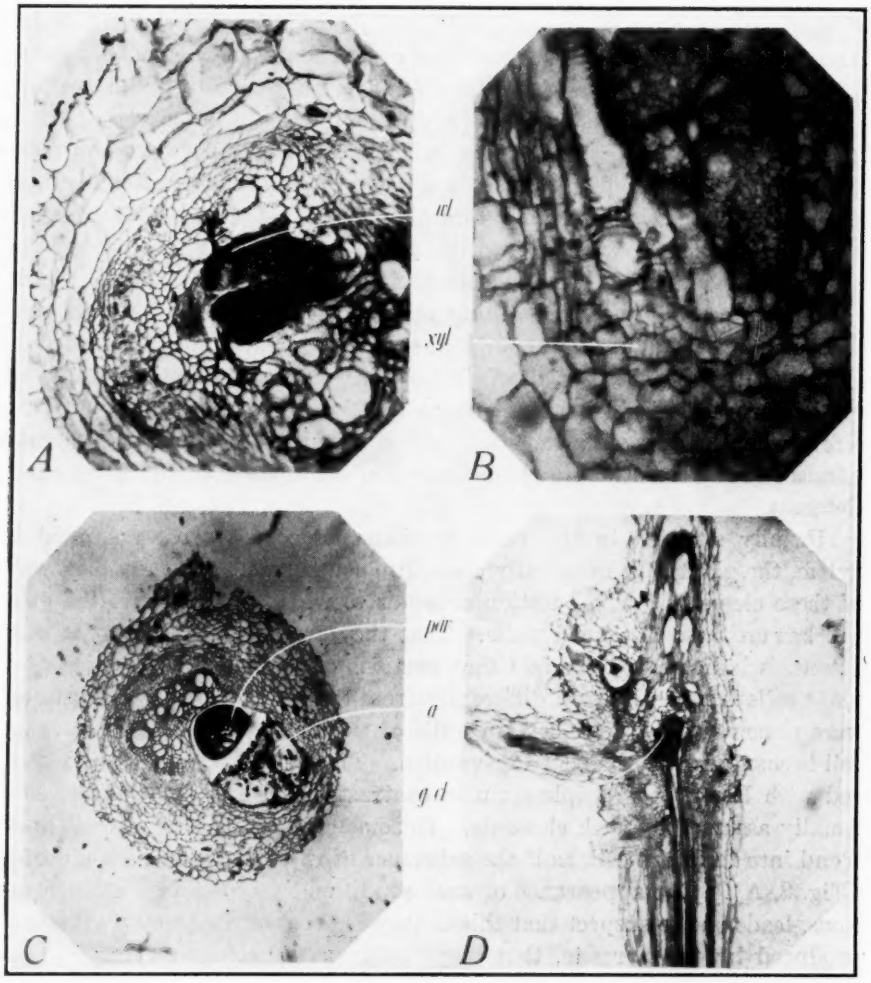

FIG. 8. A. Transverse section through 16- to 17-day gall. In places walls of xylem elements are thickened or swollen and at certain points (*wl*) appear as if undergoing dissolution and being absorbed by the giant cells. ×100. B. Longitudinal section through 10- to 11-day gall showing transformation of parenchyma into small xylem elements. Note formation of reticulate wall thickenings (*xyl*). ×280. C. Transverse section through 35- to 36-day gall passing through posterior end of parasite (*par*) and showing accumulating egg mass (*a*). ×50. D. Longitudinal section through 30- to 31-day gall. Note layer of cortical cells covering cavity in which is seen posterior end of the parasite (*par*), the head of which lies at a point near the giant cells (*g cl*). ×25.

meristem and the consequent cessation of growth. This seems to be at variance with the usual effect of parasites on many plant tissues where extensive cellular proliferation so frequently results. It is also somewhat puzzling that growth of the radicle should be suppressed, while growth of secondary roots in the region of the gall is stimulated. However, in the case of galls on the black oak, *Quercus velutina,* caused by *Andricus punctatus,* Stewart (15) notes that, soon after the gall starts, the cambium at the base of the larval chamber ceases activity, while it is stimulated to greater growth along the sides.

Another effect, manifest soon after entrance of the parasite, is hypertrophy of cortical cells and, to a less degree, cells of the endodermis and pericycle. It is interesting to note that cells of the plerome do not undergo hypertrophy except inasmuch as an increase in size coincident with initial stages of giant-cell formation may be viewed as a manifestation of this phenomenon.

The presence of the parasite stimulates cell division in the pericycle, which results in the formation of a layer of parenchyma not found in normal roots. This tendency for parenchyma to be formed in galls of all kinds is a fact emphasized by Küster (7) who writes "Vor allem ist die Tendenz zur Parenchymbildung auffällig: die Gallen sind fast durchwegs parenchymatische Gebilde." It will be recalled, however, that in the case of these nematode galls some of the parenchymatous cells eventually become differentiated into xylem elements.

Of special interest is the fact that cells of the plerome close to the head of the parasite are at first retarded in their differentiation. This is in harmony with observations on the development of many galls. Wells (18) finds, in the case of "witches-brooms" on *Celtis occidentalis,* induced by *Eriophyes* sp., that tissues in the bases of the gall branches are often markedly inhibited in their differentiation. In fact, the group of cecidia frequently designated as kataplasmas are characterized primarily by a lack of tissue differentiation. Indeed, one is reminded that some animal pathologists believe malignant tumors, in some instances at least, originate from cells that lag behind in their differentiation.

While the dissolution of cell walls in the formation of giant cells, so far as the writer is aware, has not been recorded as occurring in other types of cecidia,[2] it is probable, nevertheless, that the etiology of nematode galls does not differ, in any fundamental way, from that of galls induced by many other organisms. In the development of plant galls, the source of the stimulus, in most cases, is known. To establish the precise nature of that stimulus has been the baffling problem. There is much evidence indi-

[2] In galls produced on the apple by *Puceron langiere,* Prillieux (11) records finding *multinucleate,* parenchymatous cells.

cating that, in many instances at least, some substance produced by the parasite is the stimulating agent involved. This has been widely accepted as an altogether probable hypothesis.

While studying the structure and development of these nematode galls, one is very soon convinced that the stimulus, whatever its nature may be, emanates from the head of the parasite. The point at which initial changes take place and the subsequent arrangement and appearance of the giant cells are such as to leave little doubt in the mind of the investigator regarding the truth of this conclusion. The possibility that excretory materials provide the stimulating substance seems unlikely. Other nematodes live in plant tissues and presumably must excrete. They do not, however, induce the development of giant cells.

The frequent resemblance between callus tissue and the tissues in many galls probably has been a factor in leading plant pathologists to view critically the possible rôle of mechanical injury in inducing gall development. In many instances investigators have regarded traumatic stimuli as an important factor. Referring to galls produced by certain hemipterous insects, Cook (3) makes the unqualified statement that "the modification of the plant tissue to form the gall is purely mechanical, being a continuous effort on the part of the plant to heal the wound produced by the repeated puncturing of the cells by the insect."

In the case of galls produced by *Heterodera marioni* the possibility that mechanical injury is the exciting stimulus seems remote. All who have studied this parasite are agreed that, once its position is established, there is no evidence of mechanical injury to surrounding tissue other than that occasioned by gradual pressure due to growth and reproduction. The stylet is apparently never employed to puncture cells lying adjacent to the head. The parasite lies inert in the plant tissue moving but slightly if at all. The dissolution of cell walls and the progressive spread of the giant cells into regions a considerable distance from the parasite can scarcely be explained on the basis of traumatic stimuli. It is, of course, probable that processes are operative similar to those that usually manifest themselves when a foreign body is embedded in living tissue. The proliferation of cortical cells that tends to form an enclosing wall around the parasite may conceivably be the result of this type of stimulus.

That gall formation may be induced through the removal of substances by the parasite is discussed by Němec (10) who points out that other organisms remove substances from plant tissues without stimulating the production of giant cells or the development of galls. This investigator might have qualified his statement to the effect that some other *nematodes* habitually remove substances from the *rootlets* of plants without inducing such developments.

It is probable, therefore, that these various morphological changes in root development are brought about through the stimulating action of some substance secreted through the mouth of the parasite. All the known facts point very convincingly to such a conclusion.

SUMMARY

The galls studied were produced by experimentally infesting the radicles of tomato seedlings with the nematode *Heterodera marioni*. The age of each gall was known to within the limits of 24 hours and represented a series varying from 24 hours to 40 days. The entering larvae tend to pass between the cells. Injury to the root through cell destruction is slight. When permanently located, the head of the larva usually is in the plerome near the beginning of the region of elongation. The immediate effects on the root cells are: hypertrophy of cells in the cortical region; slight hypertrophy of cells of the pericycle and endodermis when lying near the path of the parasite; a stimulation of cell division in the pericycle; and, frequently, a suppression of cell division in the apical meristem.

During the first 48 to 60 hours, cells of the central cylinder lying in the region of the parasite's head remain undifferentiated. After about 3 days these undifferentiated cells enlarge slightly, their nuclei swell, and their walls disintegrate. The protoplasmic contents of adjacent cells coalesce to form a giant cell. Frequently these are members of rows that normally would have contributed to the formation of a vessel. The giant cell invades adjacent areas and other cells are absorbed after dissolution of cell walls. Eventually, nuclear membranes break down and giant-cell nuclei coalesce and finally disintegrate.

Division of the pericycle, stimulated by the presence of the parasite, results in a layer of small-cell parenchyma, outgrowths of which form the lateral roots that so frequently occur. Eventually, some of the innermost of these small parenchymatous cells become differentiated into xylem elements of irregular shape.

That these changes are induced through mechanical injury, through the removal of substances by the parasite or by the stimulation of excretory products, seems unlikely. It appears probable that these morphological developments in the root are due to the stimulating action of some secretion expelled through the mouth of the nematode.

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PATHOGENICITY AND VARIATION IN PHYTOPHTHORA SPECIES CAUSING HEART ROT OF PINEAPPLE PLANTS[1]

F. P. MEHRLICH

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INTRODUCTION

During the period of commercial pineapple growing in Hawaii, heart rot has been one of the principal causes of failure of young plants. The disease is present to a limited extent in almost every field during the early months of each planting.

In 1930 an extensive study of the disease and of the causal organisms was started by the writer. Its primary purpose was to find an economic control of the disease. Other phases of the study sought to relate heart rot to other diseases of the pineapple plant and of other economic and weed plants; to study the epiphytology of heart rot; to learn more of the physiology and morphology of the pathogens.

This paper presents a record of the world distribution of the disease and its local occurrence in the Hawaiian Islands; discusses the synonymies of the pathogens and presents detailed evidence establishing the synonymy of *Phytophthora cinnamomi* Rands and *Pseudopythium phytophthoron* Sideris; and considers physiologic variations within the 3 species causing heart rot in relation to their bearing on the taxonomy of the genus Phytophthora. The recorded host range of the heart-rotting species is extended and attention is called to the relationship between the disease of plants other than pineapple to the introduction and survival of heart-rotting species in pineapple fields.

The fungicidal control of heart rot has already been reported by the writer (11). Other papers will report epiphytological studies of heart rot and root failure of the pineapple plant caused by the same organisms.

Heart rot of the pineapple plant, *Ananas comosus* (Linn.) Merr., has been reported from Costa Rica (7), Jamaica (1), Puerto Rico (6), Cuba (4), Hawaii (11, 20), and Queensland, Australia (23). Its occurrence in Haiti and the Philippine Islands has been reported verbally to the writer by pineapple growers from these regions.

The rot is firm, cheese-like, and white, bordered by a characteristic brown margin (11). It destroys the younger regions of the stem and the basal portion of the central leaves.

[1] Published with approval of the Director as Technical Paper No. 51 of the Pineapple Experiment Station of the University of Hawaii.

The Heart-rotting Organisms. The organisms reported to cause heart rot have been different species, mostly in the genus Phytophthora. Ashby (1) found *P. parasitica* Dastur causing heart rot of the Ripley variety in Jamaica. Bruner (4) stated that *P. terrestris* Sherb. caused a loss of 2 per cent in a planting of the Sugar-loaf variety in Cuba. Simmonds (23) consistently isolated an undetermined species of Phytophthora from heart-rot lesions of pineapple plants in Queensland.[2] Johnson (7) states that a bacterium was isolated and determined as the cause of the disease in Costa Rica.

In Hawaii, Sideris and Paxton (20) reported *Phytophthora meadii* McRae, *Phytophthora melongenae* Sawada, and *Pseudopythium phytophthoron* to be causal organisms. *P. meadii* was said to be the most widely distributed of the three.

Recent studies of the genus Phytophthora have called attention to the essential similarity of *P. parasitica* (2, 5, 9, 21) and *P. melongenae* Sawada. Preliminary studies by the writer demonstrated the practical impossibility of separating cultures from diseased pineapple plants into *P. melongenae* on the one hand and *P. parasitica* on the other, using morphology and size of zoosporangia as criteria. It seems more desirable to consider all such isolations as a single species, which, on the basis of precedent (9, 12), is herein designated *P. parasitica* Dastur. emend Ashby. Hence, pineapple-rotting forms classified by Sideris (19) as *P. melongenae* are treated in this paper as *P. parasitica.*

A close relationship has been shown by Leonian (9) and Tucker (21) to exist between *Phytophthora palmivora* (Butl.) Butl. and *P. meadii.* So closely are the species alike that Tucker has combined them as *P. palmivora.* Leonian merged both with *P. melongenae, P. parasitica* and others under the name of *P. omnivora* de Bary (9).

The pineapple strains of Phytophthora classified by Sideris as *P. meadii* and *P. melongenae* may be separated consistently, using Tucker's key (21), as *P. palmivora* and *P. parasitica,* respectively. Therefore, in consideration of Ashby's criticism (2) of *P. omnivora* as set up by Leonian (9), the designation *P. palmivora* will be used for the heart-rotting strains classified as *P. meadii* by Sideris and for the one similar isolation by the writer.

To learn the relative importance of the several Phytophthora species in Hawaii, isolations, in the present study, were made during a period of 3 years from diseased plants occurring in a large number of representative locations on the islands of Lanai, Molokai, Maui, Oahu, and Kauai. None of the several bacteria isolated were capable of reproducing the disease. Contrary to Sideris' findings, *P. parasitica* was found to be the most widespread of the pathogens. It occurred exclusively in all of the areas sampled

[2] Determined to be *P. cinnamomi* Rands by S. F. Ashby. Reported by Ashby in a letter to the writer.

on the islands of Lanai, Molokai, Maui, and Kauai. *P. palmivora* (*P. meadii*) was found in one field only, near Pupukea, Oahu. *P. cinnamomi*, found in one field on Oahu by Sideris and reported as *Pseudopythium phytophthoron* (19), has been found in pineapple fields from Moanalua to Waimea and Opaeula, a distance of approximately 30 miles on the western slopes of the Koolau range of mountains on Oahu. It is not in pineapple fields alone, however, being found abundantly at Kipapa in virgin soil at higher elevations than fruit culture. Here it was associated with dying plants of the fern *Dicranopteris emarginata*. It has not been found on any of the other islands, although a consistent search has been made for it.

PATHOGENICITY STUDIES

Heart Rot. A number of Phytophthora species from sources other than pineapple were tested in the laboratory to determine if they might cause heart rot. A standard technique was devised for these studies. A hole 5 mm. in diameter and 1 cm. deep was out in the base of the stem of mature, cured crowns using a sterile cork borer. Into the hole were thrust malt-agar circlets bearing the appropriate organism in pure culture. Incubation was in moist chambers, fashioned from Mason jars, at 25–27° C. for 5 days.

Two distinct groups were apparent among the organisms tested: (1) those listed in table 1 that caused heart rot, and (2) those listed in table 2 that caused no disease of pineapples. The separation of strains within single species is seen to be as follows:

Phytophthora cambivora, 6 isolations negative; *P. cinnamomi,* 5 positive, 3 negative; *P. palmivora,* 3 positive, 8 negative; *P. palmivora* (*P. arecae*), 2 positive, 1 negative; *P. palmivora* (*P. faberi*), 1 positive, 2 negative; *P. palmivora* (*P. meadii*), 2 positive; *P. parasitica,* 6 positive; *P. parasitica* (*P. melongenae*), 2 positive.

Among the cultures of *Phytophthora cinnamomi* studied, there is reason to believe that the strains originally isolated by Rands and designated 13.1, 31 and 35.1 have been handled in parallel, and have received similar treatment throughout their histories. Nevertheless, the respective abilities of these to infect the pineapple plant consistently separate them. So consistent are these differences that they appear to be of the magnitude recognized in the rust fungi as *physiological strains.* Indeed, in respect to this phase of their physiology, there is a greater difference between the strains of a single species than between certain strains of different species.

It has been demonstrated that similar differences in ability to produce disease are apparent when other species and other hosts are used (8, 21). That is, within a species there are strains that cause a specific disease and others that do not. The validity of separating any species of Phytophthora from another on the basis of the differential susceptibility of a single host,

TABLE 1.—*Strains of Phytophthora species that cause heart rot symptoms in wound-inoculated crowns of pineapple*

Designation of organism	Virulence	Source		Number[a] on culture
		Host	Author	
P. cinnamomi	+		Leonian	.....
P. cinnamomi	+	Cinnamon	Rands to Drechsler	13.1 M
P. cinnamomi	++++	Cinnamon	Rands to Drechsler	35.1 B
P. cinnamomi	+	Rhododendron	White to Drechsler	x
P. cinnamomi	+++	Pineapple	Simmonds to Ashby	.....
P. palmivora (*P. arecae*)	++		Gadd to Baarn, Baarn to Leonian	1
P. palmivora (*P. arecae*)	++		Gadd to Baarn, Baarn to Tucker	54
P. palmivora (*P. faberi*)	++	Coconut in Philippines	Hartley to Leonian	75
P. palmivora (*P. meadii*)	++++	Hevea brasiliensis	Baarn to Sideris	55
P. palmivora (*P. meadii*)	++++	Pineapple	Sideris	54
P. palmivora	++	Coconut in Porto Rico	Tucker to Leonian	79
P. palmivora	++	Hevea brasiliensis	Leonian	82
P. palmivora "Cacao group"	++		Tucker from Baarn; Ashby to Baarn	57
P. parasitica (*P. melongenae*)	++++	Eggplant	Baarn to Sideris	51
P. parasitica (*P. melongenae*)	++++	Antirrhinum	Sideris	52
P. parasitica-Schwartz' strain	++++		Baarn to Sideris	44
P. parasitica-Dastur's strain	++++		Baarn to Sideris	45
P. parasitica	+++	Potato in Oklahoma	Drechsler to Tucker	P 9
P. parasitica	+++	Potato in Kentucky	Drechsler to Tucker	P10
P. parasitica	+++	Tomato	Nolla to Tucker	P19
P. parasitica (var. *Rhei* Leonian II)	++	Rhubarb	Baarn to Tucker	.....

[a] These numbers were on the cultures when received and are presumably those of the original author whose name is given opposite the number in each case.

therefore, seems very questionable. Specifically, the validity of separating the species *P. cinnamomi* and *P. cambivora* (Petri) Buis, on the basis of the differential susceptibility of potato tubers to their attack is questioned. Such a separation has been made by Tucker (21).

In the present study susceptible pineapple crowns were inoculated with 6 separate isolations of *Phytophthora cambivora* and all gave negative results. However, 3 isolations of *P. cinnamomi* also were negative and would have been classified as *P. cambivora* if pineapple rather than potato had been the criterion of species separation.

TABLE 2.—*Strains of Phytophthora species that do not cause heart rot symptoms in wound-inoculated crowns of pineapple*

Designation of organism	Source		Number on culture[a]
	Host	Author	
P. cactorum (*P. fagi*)	Beech	Baarn to Sideris	41
P. cactorum		Baarn to Sideris	57
P. cambivora		Drechsler	—
P. cambivora	Chestnut in Secondigny, France	J. Dufrenoy to Ashby	—
P. cambivora	Chestnut in England	W. R. Day to Ashby	—
P. cambivora		Petri to Baarn	
		Baarn to Leonian	4
P. cambivora		Same as above	5
P. cambivora		Petri to Baarn	
		Baarn to Tucker	53
P. cinnamomi	Cinnamon	Rands to Drechsler	31
P. cinnamomi	Chestnut in S. Europe	Petri to H. Fawcett	Fawcett's
		Fawcett to Ashby	208
P. cinnamomi	Walnut in Australia	Ashby	—
P. citrophthora		Baarn to Sideris	47
P. hydrophila[b]		Baarn to Sideris	48
P. infestans	Potato	Baarn to Sideris	49
P. palmivora (*P. arecae*)		Leonian	2
P. palmivora (*P. faberi*)		Leonian	76
P. palmivora (*P. faberi*)		Leonian	77
P. palmivora[c] "Cacao group"	Papaya	Sideris	43
P. palmivora[c] "Cacao group"	Ricinus	Sideris	53
P. palmivora	Borassus in India	Hartley to Leonian	80
P. palmivora		Baarn to Leonian	78
P. palmivora	Grapefruit in Puerto Rico	Tucker to Leonian	81
P. palmivora		Ashby to Baarn	
		Baarn to Leonian	83
P. palmivora "Rubber group"	Coconut in Mauritius	Ashby	—
P. palmivora "Rubber group"	Coconut in Puerto Rico	Tucker	55
P. palmivora "Cacao group"	Coconut in S. India	Ashby	—
P. palmivora "Cacao group"	Palmyra palm in India	Ashby	—
P. parasitica var. *nicotiana*	Tobacco	Baarn to Sideris	—
P. richardiae		Baarn to Sideris	42
P. parasitica (*P. terrestris*)		Baarn to Sideris	56
P. sp.[d]	*Ficus carica*	Sideris	58

[a] These numbers were on the cultures when received and are presumably those of the original author whose name is given opposite the number in each case.

[b] This species is included in *P. omnivora* de Bary by Leonian and in *P. capsici* Leonian by Tucker.

[c] Subcultures of these two organisms were distributed by Sideris under the name *P. monoana* n. sp. but a description was not published. S. F. Ashby, in a letter to the writer, stated that number 43 is a typical strain of *P. palmivora* in the "Cacao group" of that species. C. M. Tucker, also, independently, made a similar designation.

[d] This culture was received from Sideris bearing the name *P. symmetrica* n. sp. A description of it has not been published and its identity has not been determined.

The writer has shown that bud rot of the palm and heart rot of the pineapple plant may be caused by the same strains of Phytophthora (Table 1, organisms numbered 1, 54, 55, 75, 79). Since this is true it is regarded as significant that both diseases occur in proximity on at least 2 of the islands of the Hawaiian group. For a comparison of these 2 diseases, see the references cited as numbers 11, 18 at the end of this paper.

It also has been demonstrated that heart rot of the pineapple plant is caused by the same pathogens that cause: (1) foliage and fruit rot of the rubber tree, *Hevea brasiliensis;* (2) cankers of the cinnamon tree, *Cinnamomum burmanni;* (3) canker of *Rhododendron* sp.; (4) foliage and stem blight of the tomato, *Lycopersicon esculentum;* (5) leaf blight and tuber rot of the potato, *Solanum tuberosum;* and (6) crown rot of rhubarb, *Rheum rhaponticum.*

This suggests the means by which the heart-rotting organisms may have been introduced into the islands. It also indicates the desirability of restricting the growth of certain vegetables where the pineapple disease is prevalent.

Green-fruit rot:

During February, 1932, pineapple fruits of four stages of development were inoculated with local isolations of *Phytophthora cinnamomi*: A, inflorescences on which the last flowers had just withered; B, half-grown green fruits; C, full-grown green fruits; and D, yellow ripe fruits. An agar circlet containing the organism was placed over needle punctures made in a single eye, or on the cut stem base. *P. cinnamomi* aggressively rotted every inflorescence, rapidly extending the lesions. Half-grown fruits also were invaded, but the rate of advance of the fungus was slow. No full-grown fruit, either green or yellow, rotted in the several tests. All tissues of the invaded fruits appeared susceptible, the core rotting as well as other parts.

A confirmatory test was conducted during February, 1934. Fruits corresponding to the first 3 classes in the earlier test were selected in pairs, each pair being nearly uniform in weight and appearance. One fruit of each pair was inoculated with culture 60 of *Phytophthora cinnamomi* by placing an agar culture in a wound made with a 5 mm. cork borer. The crowns and peduncles were then removed from the other fruits. They were cut so that separate determinations of the pH of juice, expressed by hand, could be made on the shell, the core, and the upper and lower halves of the flesh. Table 3 shows the weights and pH of these fruits. Following an incubation of 15 days at 25–27° C. the inoculated fruit of each pair was examined. All inoculations made in inflorescences of class A were positive regardless of their position on the fruit. All fruits of class B likewise showed disease; inoculations had been made in the upper and lower halves

TABLE 3.—*The weight and hydrogen-ion concentration of immature fruits inoculated with Phytophthora cinnamomi*

Maturity of fruit[a]	Weight of fruit in grams		Hydrogen-ion concentration of fruit parts[b]							
			Flesh				Core		Shell	
			Upper half		Lower half					
	Range	Av.	Range	Av.	Range	Av.	Range	Av.	Range	Av.
A	450– 520	485	4.72–5.08[c]	4.96						
B	1380–1580	1458	3.79–4.00	3.87	3.48–3.94	3.75	3.59–4.23	3.98	4.21–4.64	4.49
C	2640–2960	2770	2.75–3.29	2.94	2.41–2.99	2.66	2.75–3.01	2.91	3.58–3.71	3.65

[a] The fruit classes are as follows: A, inflorescences with last flowers just withered; B, half grown green fruits; C, fully grown green fruits.

[b] Determinations made with hydrogen electrode.

[c] All parts of the inflorescences of class A were composited.

of the fruit as well as in the core at the base of the fruit. In all cases lesions from inoculations in the upper regions of the fruit expanded more rapidly than similar inoculations in the lower regions.

No inoculations in any fruit of class C produced a lesion, thus verifying the earlier observations. Figure 1 shows a typical fruit of class B. Observe the difference in size of the lesions, and the distribution of disease through all of the tissues of the fruit, including the core and the shell. Note the characteristic brown margin bordering the grayish white, firm, rotted areas.

FIG. 1. Rot of green pineapple fruit caused by *Phytophthora cinnamomi*. Photographed 15 days after inoculation. Note the dull white, dry rotted areas surrounded by the characteristic brown margins. Observe that the basally situated lesion is the smaller one.

A difference also is noticeable in the color of the healthy and diseased shell areas: the infected areas are brownish and may be covered with white mycelium. In one case this mycelium had grown over the superficial tissues of the shell to the crown of the fruit, where typical heart rot developed. The lesion, however, had not expanded so rapidly through the internal tissues.

Similar results were obtained with *Phytophthora parasitica* (*P. melongenae*), but no disease was produced by *P. palmivora* (*P. meadii*). Young inflorescences of a series of fruits susceptible to the other species under similar conditions, were repeatedly inoculated with *P. meadii* by both the writer and C. H. Spiegelberg,[3] but disease developed in no single instance.

Waldron, in 1928, first described a rot of green pineapple fruits occurring naturally on the islands of Kauai and Oahu. *Phytophthora meadii* was reported[4] to be the pathogen and disease was reported to have been experimentally reproduced using this organism. The symptoms she described are similar to those illustrated above in figure 1, produced in the present study with *P. cinnamomi* and *P. parasitica* only.

No disease was found in the field from 1928 until January, 1933, when a single specimen was found on Kauai by M. B. Linford. It is possible that green fruit rot caused by the heart-rotting Phytophthoras may again cause significant losses.

Waldron[4] reported: "Susceptibility to Phytophthora rot appears to depend on the lower acidity of younger fruits, which have pH values ranging between 5.4 to 4.4, whereas those of fully developed fruits, which are resistant to the disease, lie between 4.4 and 3.4."

The results of the present study seem to substantiate a correlation between pH and development of the disease: Not only did disease fail to develop in the more acid fruits, but, in fruits where lesions were evident, the disease had extended most rapidly through the tissues of lowest acidity.

Weed and Green-manure Plants.—As a phase of epiphytological studies carried on during a period of 3 years, a number of weeds common in cultivated and fallow pineapple fields were tested. Also the commoner green-manure plants were inoculated. Table 4 summarizes the results.

The ability of these Phytophthoras to infect wounded weed or green-manure plants, while perhaps of little taxonomic significance (22), is nevertheless, indicative of material suitable for the growth of the heart-rotting organisms in the absence of the pineapple, and thus constitutes a factor in survival of the pathogenes in the field. Plowing and disking periodically produce such wounded materials in abundance, often during weather suitable for growth of these fungi. From this standpoint it is significant that the 3 common green manure plants, Crotalaria, Mucuna, and Cajanus, are

[3] Unpublished notes, 1930.

[4] Private communication to the local pineapple industry, 1928 and 1930.

TABLE 4.—*Weeds and green-manure plants tested for susceptibility to pineapple heart-rotting organisms*

Plant tested and part used	Organisms tested and inoculation type													
	44[a]		52		54		60		384		443		303	
	W	NW	W	NW	W	NW	W	NW	W	NW	W	NW	W	NW[b]
Carica papaya L. seedlings	+	−	−	−	+	−	−	−	−	−	−	−	−	−
Crotalaria incana L. seedlings	+	+	+	−	+	+	−	−	−	−	−	−	−	−
Macuna gigantea (Willd.) DC. seedlings	+	−	+	−	+	−	Sl. +	−	−	−	−	−		
Cajanus cajan (L.) Millsp. seedlings	+	−	+	−	+	+	+	−	+	−				
Lycopersicon esculentum Mill. seedlings	+	+	+	−	+	−	Sl. +	−	Sl. +	−	−	−	Sl. +	−
Melia azedarach L. seedlings	−	−					−	−			−	−		
Melia azedarach L. roots	−	−												
Melia azedarach green fruits			+	−										
Ricinus communis L. seedlings	+	+	Sl. +	−	Sl. +	−	−	−	−	−	−	−	−	−
Ricinus communis L. radicles			+	+	+	+	+	+						
Emelia sonchifolia DC. seedlings	−	−	−	−	−	−	−	−	−	−	−	−	−	−
Richardsonia scabra St. Hil. seedlings	+	+	+	+	+	+	− to Sl. +	−	−	−	−	−	+	−
Ageratum conyzoides L. seedlings	−	−	−	−	−	−	−	−	−	−	−	−		
Bidens pilosa L. seedlings	+	Sl. +	−	−	−	−	−	−	−	−	−	−		
Erigeron canadensis seedlings	−	−	−	−	−	−	−	−	−	−	−	−		
Dicranopteris emarginata (Brack) W. J. Robinson, young fronds											−	−		

[a] The numbers refer to Phytophthora species: 44—*P. parasitica;* 52—*P. parasitica* (*P. melongenae*); 54—*P. palmivora* (*P. meadii*); 60, 384, 445—Hawaiian isolations of *P. cinnamomi;* 303—*P. cinnamomi* from Leonian.

[b] W indicates inoculation made following wounding of host; NW indicates no wounding.

susceptible to more than one of the heart-rotting species. Two of the widely distributed weeds, Ricinus and Richardsonia, are likewise very susceptible, giving a basis for the observation that where Ricinus occurs in abundance, heart rot also occurs.

Economic Vegetable and Flowering Plants. In an effort to prove the synonymy of *Phytophthora cinnamomi* Rands and *Pseudopythium phytophthoron* Sideris, the pathogenicities of authentic strains of *Phytophthora cinnamomi* were compared with Sideris' organsm. Plant parts used by Tucker in his studies were inoculated with 3 isolations of Pseudopythium and with 3 isolations that served as the type for Rands' description of *P. cinnamomi.*

The pathogenicities of these 6 cultures are compared in Table 5 with Tucker's cultures 242 and 244 of *Phytophthora cinnamomi* and a single culture of *P. cambivora.* The 3 isolations of Pseudopythium all agree favorably with the strains of *Phythophthora cinnamomi.* Two of Rands' original isolations cause heart rot of pineapple crowns. One of these, 35.1, compares favorably in virulence with Hawaiian strains from pineapple. Other strains obtained from Leonian and Drechsler are slightly pathogenic (Table 1).

The finding of *Phytophthora cinnamomi* as a cause of heart rot in Queensland, Australia, (isolations of Simmonds identified by S. F. Ashby) strengthens the prior conclusion by the writer that Pseudopythium is synonymous with this species (10). To confirm additionally the identity of *Phytophthora cinnamomi* and Pseudopythium, 5 local isolations from pineapple were sent to R. P. White for comparison with his isolations from Rhododendron.[5] All were pathogenic on Rhododendron and agreed as to type of canker produced on the stems and as to rate of invasion. White's cultures from Rhododendron were found to be only mildly pathogenic on pineapple, as shown in table 1.

CULTURAL STUDIES

Physiology. Tucker, who studied the effect of temperature on the rate of growth of a number of Phytophthora species, found that 2 strains of *P. cinnamomi* failed to develop above 35° C. while 3 other isolations failed to develop above 32.5° C. The optimum temperature for growth he reported lies between 20° and 25° C.

Table 6 presents the results of similar studies made with a subculture of the type of Sideris' Pseudopythium, organism 60. Petri dish plates (100 mm.) containing 12 cc. of Difco corn meal agar (pH 6.4) were inoculated with 5 mm. discs of similar medium cut from the marginal growth of a 72-hour culture. These were incubated in triplicate at the several

[5] Results reported by letter to the writer, April 30, 1932.

TABLE 5.—*A comparison of the pathogenicities of cultures of Pseudopythium phytophthoron Sideris with Phytophthora cinnamomi Rands and Phytophthora cambivora*[a]

Host plant and part used	Organisms used and pathogenicity								
	Pseudopythium phytophthoron			Phytophthora cinnamomi					Phytophthora cambivora
	60	384	443	13.1	31	35.1	242	244	
Papaya seedlings	−	−	− to V. sl. +	(−)	(−)	(−)	(−)	(−)	−
Apple fruits	+	+	+	(+)	(+)	(+)	(+)	(+)	+
Potato tuber	++	++	++	(++)	(+)	(+)	(++)	(++)	−
Tomato fruits	+	+	+	(+)	(−)	(+)	(+)	(+)	−
Egg plant fruits				(−)	(−)	(−)	(−)	(−)	−
Ricinus seedlings	−	−	−	(−)	(−)	(−)	(−)	(−)	−
Tomato seedlings	−	−	−	(−)	(−)	(−)	(−)	(−)	−
Egg plant seedlings	−	−	−	(−)	(−)	(−)	(−)	(−)	−
Bryophyllum leaves	−	−	−	(−)	(−)	(−)	(−)	(−)	−
Pineapple crowns	++	++	++	+	−	++			−

[a] Cultures numbered 60, 384, 443 are local isolations from pineapple. Cultures 13.1, 31, and 35.1 are strains of *P. cinnamomi* isolated by Rands from *Cinnamomum burmanni* in Sumatra. Cultures 242 and 244 are Tucker's isolations from *Persea persea* in Puerto Rico. The data in parenthesis are taken from Tucker's paper (21) but were verified for organisms 13.1, 31, 35.1.

TABLE 6.—*The mean diameter of mycelial growth in plate cultures of Phytophthora cinnamomi (Pseudopythium phytophthoron) strain 60 on cornmeal agar (pH 6.4) incubated 96 hours at various temperatures*

Temperature degree C.	Mean diameter in millimeters	
	Range of 3 plates	Average
12.8 ± 0.5	4.5 – 4.5	4.5
15.6 ± 0.5	17.5 – 23.0	20.1
18.3 ± 0.5	27.5 – 27.5	27.5
21.1 ± 0.5	35.5 – 38.5	38.5
23.9 ± 0.5	36.5 – 43.5	40.0
25.0 ± 1.0	52.5 – 55.5	54.0
26.7 ± 0.5	44.5 – 44.5	44.5
27.8 ± 0.5	33.5 – 34.5	34.0
30.0 ± 1.0	34.5 – 39.5	36.5
32.5 ± 1.0		No growth[a]
35.0 ± 1.0		" "
37.5 ± 1.0		" "
40.0 ± 1.0		" "

[a] Slight growth less than 1 mm. occurred in one plate.

temperatures for 96 hours, and then the diameter of growth was determined (colony diameter minus 5 mm.). The fluctuation between plates, as well as the average for each series, is given. The optimum temperature for growth is 25° C.; the maximum is 30° C.; the minimum near 10° C. The cardinal temperatures of other local isolations, 384, 407, and 443, are identical with those reported for strain 60. Hence, local isolations compare favorably in temperature relationships with Rands' organism 35.1, reported as number 174 in Tucker's paper (21).

While the cardinal temperatures for the several strains of *P. cinnamomi*

TABLE 7.—*The mean diameter of mycelial growth of strains of P. cinnamomi grown on malt agar for 72 hours*

Strain[a]	Mean colony diameter mm.[b]		
	25° C.	30° C.	35° C.
443	49.0	49.5	0
35.1	49.5	59.0	0
Leonian's	63.0	42.3	0
13.1	38.5	39.0	0

[a] For source of cultures see table 1.

[b] The mean value shown is for five plates at each temperature.

TABLE 8.—*The mean diameter of mycelial growth of strains of heart rotting Phytophthora sp. grown on papaya agar for 72 hours at 25–27° C.*

Organism	Strain	Source	Mean colony diameter mm.[a]
P. cinnamomi	60	Waialua, Oahu	66.0
"	407	Helemano, Oahu	60.5
P. palmivora	54	C.B.S. Baarn	54.0
"	55	" "	43.3
P. parasitica	417	Pupukea, Oahu	38.2
"	415	Hanapepe, Kauai	37.3
"	414	Kualapuu, Molokai	34.5
"	401	Kipu, Molokai	32.5
"	393	Island of Lanai	28.5
"	51	C.B.S. Baarn	25.8

[a] The mean value shown is for 5 plates of each organism.

appear to be very similar, their rates of growth are dissimilar throughout a range of temperatures. As an example, contrast the colony diameters of strains 35.1 and 13.1 shown in table 7. It is important to note, moreover, that the rate of growth of these organisms on artificial media is not necessarily related to their virulence, shown in table 1. Leonian's strain and strain 13.1 advance at about an equal rate through pineapple tissue, but their rates of growth on agar are very different (Table 7).

Similar differences occur between the 3 species that cause heart rot: *P. cinnamomi, P. palmivora,* and *P. parasitica.* Papaya (20) and malt

TABLE 9.—*The mean diameter of mycelial growth of P. palmivora and P. parasitica in plate cultures on corn-meal agar (pH 6.4) incubated 96 hours at various temperatures*

Temperature degrees Centigrade	Mean colony diameter in mm.[a]	
	P. palmivora Strain 55	*P. parasitica* Strain 414
12.8 ± 0.5	30.5	4.0
15.6 ± 0.5	41.0	22.0
18.3 ± 0.5	58.0	32.0
21.1 ± 0.5	66.0	38.0
23.9 ± 0.5	66.0	43.7
26.7 ± 0.5	63.2	46.0
30.0 ± 1.0	.....	43.0
32.5 ± 1.0	25.0	40.0
35.0 ± 1.0	0	20.0
37.5 ± 1.0	0	8.0
40.0 ± 1.0	0	0

[a] The mean value shown is for 5 plates at each temperature.

agars (12) seem to accentuate differences in rate of growth of the 3 species, as well as the strains of a single species, as shown in table 8. On corn-meal agar the differences are consistently in the same direction, but of a lesser magnitude. No similar differences have been noted in the rate at which heart rot lesions are extended. These observations suggest differences in the enzyme activities of the several strains.

Table 9 shows the rates of growth of typical strains of *Phytophthora palmivora* and *P. parasitica* at various temperatures. These growth curves should be compared with that of *P. cinnamomi* shown in table 6. *P. cinnamomi* is distributed in the cooler upland areas of Oahu, only, while *P. parasitica* is found also in the hotter locations, near the sea level on 3 of the islands sampled. It apparently is more tolerant of higher temperatures.

Table 9 shows that no growth of strain 55 of *Phytophthora palmivora* occurred at 35° C., but that strain 414 of *P. parasitica* did grow at this temperature. This difference is in agreement with the observations of Tucker who regards it to be of diagnostic value in separating the two species (21). For the large number of strains of both species with which Tucker worked this temperature relationship appeared to be constant, but for isolations of *P. parasitica* from pineapple plants it does not seem to hold. Table 10 is a summary of the behavior of a number of representative isolates from

TABLE 10.—*The behavior of heart rotting strains of Phytophthora incubated in plate cultures of Difco corn-meal agar (pH 6.4) for 4 days at 35° C.*

Organism number	Source	Growth at 35° C.[a]	Growth at 25° C. following incubation at 35° C.[b]
72	Helemano, Oahu	–	+
77	Kilauea, Kauai	–	–
395	Maunaloa, Molokai	–	+
398	Brodie, Oahu	–	–
399	Helemano, Oahu	–	+
400	Kualapuu, Molokai	–	+
401	Kipu, Molokai	+	+
417	Pupukea, Oahu	+	+
AC 34	Miki, Lanai	+	+
414	Kipu, Molokai	+	+

[a] The sign + indicates growth, the sign – indicates no growth.

[b] Incubation at 25° C. followed incubation of 4 days at 35° C.

heart-rot lesions. If growth at 35° C. on corn-meal agar were the criterion for separating the species, the first 6 isolates would be classified as *P. palmivora* and the last 4 as *P. parasitica*. Morphologically, however, all of the organisms appear to be strains of *P. parasitica* Dast. emend Ashby. The sporangia, chlamydospores, and mycelial characters more nearly agree with

typical strains of this species than with *P. palmivora.* Hence, the growth or lack of growth of Phytophthora strains at 35° C. on corn-meal agar seems an unsuitable basis for separating *P. palmivora* and *P. parasitica.*

Morphology. A comparative examination of the morphology of Hawaiian isolations 60, 384, and 443 of the organism named *Pseudopythium phytophthoron* by Sideris with strains 31 and 35.1 of *Phytophthora cinnamomi* Rands isolated by Rands revealed no significant differences in the

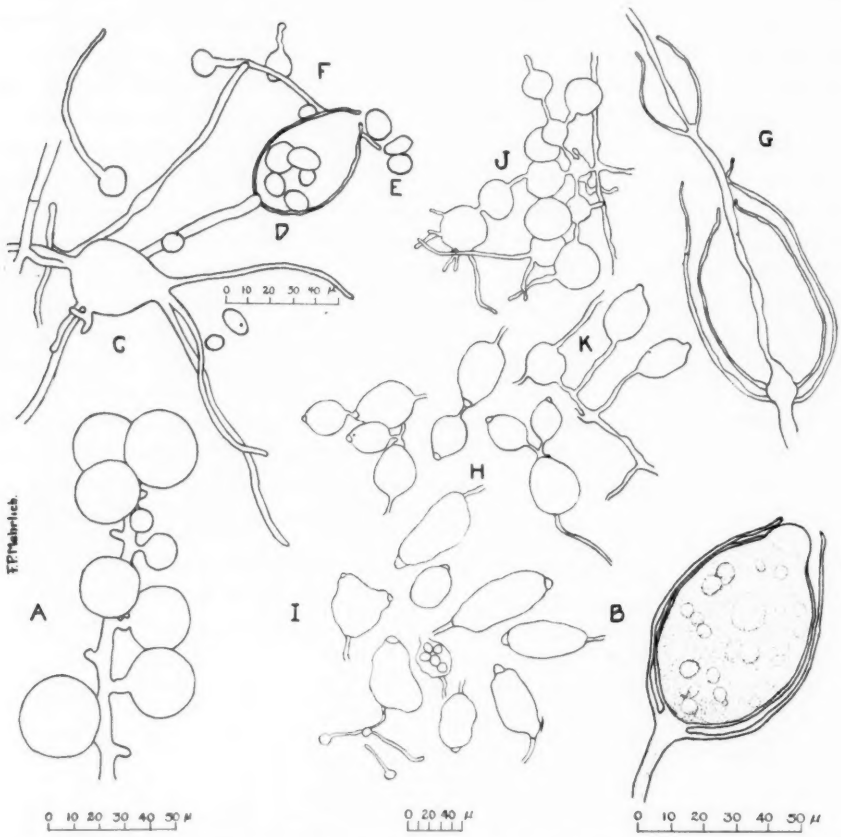

FIG. 2. Asexual reproductive structures of Hawaiian isolations of *Phytophthora cinnamomi* Rands (*Pseudopythium phytophthoron* Sideris) drawn with camera lucida. A. Cluster of chlamydospores. B. Full-grown papillate, primary sporangium within the empty walls of a dehisced sporangium. C. Primary sporangium germinating by germ tubes. D. Dehisced sporangium containing encysted zoospores. E. Zoospores shortly after being discharged from sporangium. F. Zoospores germinating by vegetative germ tubes. G. Sporangia showing proliferation. H. Sporangia germinating by the production of secondary sporangia. I. Primary sporangia of various shapes. J. Hyphal vesicles resembling chlamydospores produced in M/100 KNO_3. K. Hypha bearing both sporangia and a vesicle.

size of mycelium, "chlamydospores," hyphal vesicles, or conidia when grown under similar conditions. The diameter of hyphae of the Hawaiian isolations average 10 microns; (on Difco corn-meal agar, pH 6.4). Lateral branches are only slightly smaller, rarely being less than 5 microns.

The "chlamydospores" of the Hawaiian isolations may be wholly spherical, varying in diameter from 10 to 36 μ, averaging 20 μ at the bottom of Petri plate cultures and 25 μ on aerial mycelia. They may intergrade in both form and dimensions with the abundant hyphal vesicles of irregular shape and size, or may occur in grape-like clusters, or be disposed singly and terminally on short lateral branches (Fig. 2). The "chlamydospores" germinate by germ tubes, 1 to 6 or more. The germ tubes in liquid cultures may give rise to vegetative hyphae or rarely produce zoosporangia. The ellipsoidal zoosporangia are produced in very dilute liquid media only, borne on short (50–75 μ) or long (75–790 μ) simple or branched sporangiophores. They are irregular in size, shape, and proportions and are characterized by successive proliferation. The methods to induce zoospore formation are reported elsewhere (13). Produced by those methods, the majority of the zoosporangia of Rands' cultures and the Hawaiian isolations show conspicuous papillae (Fig. 2). These contrast with the blunt, nonpapillate sporangia reported by all other workers. Of 225 sporangia counted in a nonsterile soil solution (pH 4.5), 183 were papillate. Distorted sporangia showing more than one papilla are not rare. Dehiscence occurs at the base of the papilla, the opening ordinarily being less than the diameter of the zoospores. Zoospores, although fully formed, may not be discharged. In such cases they germinate within the sporangium and send the germ tubes through its walls. Zoospores may not be formed at all, the conidia giving rise to one or more hyphae or to secondary conidia. Characteristically 2 secondary conidia are formed, although 1 to 3 have been observed to originate from a single primary conidium. The zoospores are active only a short time under conditions observed, germination at 20° C. taking place between 4 and 5 hours after the spores are liberated. Various of these stages are illustrated in figure 2, by camera-lucida drawings of local isolation 60 of *Phytophthora cinnamomi* (*Pseudopythium phytophthoron*).

In table 11 the extensive variability of zoosporangia is demonstrated by isolation 384 in a soil extract solution of approximately pH 4.5. The ratio of sporangial length to width varied between 1.33 and 2.22; average 1.77. Zoospores liberated either from the larger or smaller sporangia show a range of sizes, a few being either much larger or smaller than the average. The number of spores liberated is directly correlated with the size of the sporangium. Twenty-five zoospores selected at random from sporangia recorded in table 11 measured in microns after coming to rest: 3.8–10 × 8.8–13.9, average 6.4 × 11.6.

TABLE 11.—*The size in microns of representative zoosporangia of P. cinnamomi, strain 384, produced in soil-extract solution pH 4.5*

Length	Width	22.68	27.27	27.72	30.24	30.48	32.76	33.02	35.28	35.56	37.80	38.10	40.64	43.18	45.72	47.88	48.26	50.80	53.34	55.88	Total
47.88					1				1												2
49.14					1																1
50.40				1	1		1		1			1									4
50.80																					1
52.92					1		2				1										3
55.44							1			1		1	1								2
55.88			1			1		1													5
60.48										1		1	2								1
60.96							1														4
61.70									1												1
65.52		1											2	1	1						2
66.04										1											4
68.58													4	2							1
71.12														1							6
73.66						1							2	1	1		1				2
76.20																1					5
78.74							1														1
80.56																1					1
80.72													7		2		2	1			1
81.28															2		1	1			12
86.36																	1				4
88.90																	1		1	1	1
91.44																	1				3
93.98														1				1			1
96.52																				1	2
99.06															1					1	2
101.60																					1
Total		1	1	1	4	2	6	1	3	3	1	3	18	6	7	2	7	3	1	3	73

Attempts to produce oospores have been unsuccessful, although media and temperatures used by others to produce oospores in other strains have been duplicated. Local isolations paired variously with other local isolations, as well as with Rands' strains 13.1, 35.1 and Simmonds' strain from pineapple in Australia, have all given negative results.[6] Pairings also were made with several strains of *Ph. cambivora* but were likewise negative.

It is believed that the essential morphology of the local isolations 60, 384, and 443, described above, is sufficiently similar to authentic strains of *Phytophthora cinnamomi* Rands to indicate the synonymy of this species and *Pseudopythium phytophthoron* Sideris.

The 6 strains of *Phytophthora cambivora* (Petri) Buis. studied comparatively with 6 strains of *P. cinnamomi* showed a close similarity to *P. cinnamomi.* The differences between the species were very minor ones in amount of growth of aerial mycelium and in abundance of "chlamydospores" produced on agar plates or slants. These differences are suggestive of strain or, at best, of varietal differences of a single species, only.

Between the size and shape of zoosporangia or size and character of oogonia produced by the 2 species, there appear to be no significant differences recorded by other workers (2, 3, 9, 14, 15, 16, 17, 21) (Footnote 6, p. 15). Throughout the range of usual physiological reactions and over a considerable host range the 2 species are similar (21). It is suggested, therefore, that *P. cambivora* (Petri) Buis. and *P. cinnamomi* Rands be combined as a single species.

SUMMARY

The record of the world distribution of pineapple heart rot is extended and brought up-to-date. The results of a 3-year study on the distribution in the Hawaiian Islands of the 3 local causal organisms are given, indicating the presence of *Phytophthora parasitica* on all of the islands sampled, *P. cinnamomi* over a distance of 30 miles on one drainage slope of Oahu only, and *P. palmivora* in a limited area on Oahu, only. These findings are in contrast with the distribution reported for the species in the islands by Sideris and Paxton (20).

Synonymies of the heart-rotting pathogens are discussed, and detailed evidence is presented for considering *Pseudopythium phytophthoron* Sideris as a strain of *Phytophthora cinnamomi* Rands.

[6] In a communication to the writer S. F. Ashby reported the following dimensions for the sexual organs of Simmonds' strain of *P. cinnamomi*, in microns: Oogonia (mean of 62) 45.4, range 37–58; oospores (mean of 19) 38.5, range 33–49; antheridia mostly 16–29 × 16–25, but occasionally transversely septate and elongate, up to 33 × 21. The oogonia were reddish brown; the antheridia amphigynous. Only one-third of the oogonia contained differentiated oospores. They were formed at the base of a Quaker oats slant after 45 days at a mean temperature of about 16° C.

Variations are demonstrated between cultures within the species *P. cinnamomi*, *P. palmivora*, and *P. parasitica*, of a nature recognized in the rust fungi as *physiological strains*. The recognition of these differences casts doubt on the validity of separating species of Phytophthora on the basis of the differential susceptibility of a single host.

The validity of separating *P. cinnamomi* Rands from *P. cambivora* (Petri) Buis. by the differential production of rot of potato tubers (21) is questioned. Since the morphology of the 2 species is essentially similar, it is suggested that they be combined as the single species *P. cambivora*, in which strain differences may be recognized.

Data are presented showing that a number of *P. parasitica* isolates from pineapple heart rot in Hawaii are more sensitive to high temperatures than strains studied by Tucker (21). Since these organisms do not grow on cornmeal agar at 35° C., this criterion of Tucker's for separating *P. parasitica* from *P. palmivora* does not seem reliable.

New host-range relationships of the pineapple pathogens are discussed, including an experimentally produced green pineapple fruit rot. The relationship of Rhododendron canker, bud rot of palms, foliage and fruit rot of the rubber tree, canker of the cinnamon tree, and rots of common weed, truck-crop and green-manure plants of pineapple fields to pineapple heart rot is reported.

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THE IDENTITY OF RASPBERRY MOSAICS[1]

L. M. Cooley

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Different opinions exist as to the number of viruses concerned in the etiology of mosaic diseases of raspberries in eastern North America. Because of this much confusion and uncertainty has ensued in the general understanding about these maladies.

While the writer was investigating the nature and identity of the so-called "mild mosaic" in the Columbian variety of the hybrid or purple raspberry (*Rubus neglectus* Peck), results appeared that had a direct bearing on the larger question of the number of raspberry mosaic viruses. It is hoped that presentation of this evidence and a discussion of the experiences and opinions other investigators have recorded in the literature will simplify and clarify concepts of the raspberry mosaics.

NATURE AND IDENTITY OF THE "MILD MOSAIC" IN COLUMBIAN RASPBERRIES

Survey of the Problem

The variety Columbian is the only purple raspberry of commercial importance, but it is grown extensively in some sections. Foliage of plants of this variety usually exhibits a mild mottling, accompanied by little or no injury. So common is the occurrence of this mottling in Columbian stock that it has come to be regarded by growers as a varietal characteristic. This mottling is usually most evident and general on the first leaves produced by fruit-spur growth in the spring. It also occurs noticeably on the first leaves of the new shoots (turions) and may become evident sporadically on foliage produced later. Leaves that are grown during or following cool weather especially are apt to show the mottled effect.

Such symptoms when found in black raspberries have been designated "mild mosaic" by Dodge and Wilcox (7) and by Bennett (1). However, opinions in literature as to the nature and relationships of this mild mottling in hybrid raspberries have been indefinite and variable and largely a matter of conjecture.

In 1927, Bennett (1) included in a discussion of red (raspberry) mosaic "two rather distinct types of mottling" occurring in Columbians. The milder of his two types is apparently identical with the "mild mosaic" of Columbian raspberries dealt with herein. But no statements of inoculation trials on indicator black raspberry plants were given to support placement of this disease as red (raspberry) mosaic. In later contributions,

[1] Approved by the Director of the New York State Agricultural Experiment Station as Journal Paper No. 86, March 14, 1935.

Bennett (2, 4) classified this mild mottling of purple raspberries with "mild mosaic" as affecting all species of raspberries.

Rankin's published comments reflect an evolution in ideas pertaining to this question. In 1924, he apparently did not recognize this mild mottling as a mosaic and stated (8, p. 1): "The purple variety Columbian is practically free of mosaic in the southwestern part of Erie county and in Chautauqua county [New York]." In 1927, he published these statements (9, p. 9): " 'Mild mosaic' of raspberries is of doubtful nature and of little or no commercial importance, although widely prevalent. . . . It is suspected that 'mild mosaic' is due to a temporary infestation of red mites which cause this injury of feeding on the unfolding leaves. . . . Many standard purple varieties seem to be more favorable hosts for the rapid multiplication of the red mite and the typical injury is common on such varieties." In March, 1931, (11, p. 5, 6) he indicated a suspicion that the "mild mosaic" of Columbians may be caused by the red (raspberry)-mosaic virus in the following assertions: "It seems doubtful if there exists a distinct virus disease that should be called mild mosaic. This statement includes the so-called 'mild mosaic' in purple varieties in which it is believed that all types of virus mottling are referable to either red (raspberry) mosaic or to yellow mosaic."

Cooley and Rankin (6, p. 6), in December, 1931, reported that, "In some of the experimental plantings the source of red [raspberry] mosaic invasion seemed to be traceable to nearby cultivated Columbian plantings, showing the 'mild mosaic' typical of this variety." These findings of field study were considered proof that the mild mosaic in purple raspberries was identical with red (raspberry)-mosaic in black raspberries. In 1933, Rankin (13, p. 30) stated that, "All Columbian stock has been uniformly affected with red [raspberry] mosaic for several years."

It is singular that no experimental inoculation tests made for the specific purpose of determining the nature and identity of this malady have been reported. Bennett (5), in studies on the constancy of degree of raspberry-mosaic symptoms, reported that 11 of 25 healthy black-raspberry plants (Cumberland variety) became affected with *mild mottling only* when aphids of the species *Amhorophora rubi* were transferred to them from mildly mottled Columbians. This appears inconsistent with his classification in 1927 of the mild mottling of Columbian purple raspberries as red (raspberry) mosaic, at which time his concept (1) of that disease on black raspberries was of a rather severe type.

In 1931, a project on the maintenance of healthy black raspberry plantings was begun by the writer in southern Erie County in western New York. Columbian plantings are numerous in this district. Two types of mottling are present in them, that of the "mild mosaic" sort is universal; that of yellow mosaic is found only occasionally. It was deemed

essential to find out experimentally whether the mild type expressed the presence of a contagious virus, and, if so, to determine its identity and evaluate its menace to the healthy black raspberries.

Field Experiments and Observations

Etiology of Columbian "Mild Mosaic." During the growing seasons of 1932 and 1933, 5 series of transmission tests on black raspberries were carried out under the conditions and with the results given in table 1.

TABLE 1.—*The tranmission of "mild mosaic" of Columbian hybrid raspberry plants to healthy Cumberland and Plum Farmer black raspberry plants, using Amphorophora rubi[a] as the vector.*

Series and date of inoculation	Variety	Location and conditions	Treatment	No. of plants	Infections red (raspberry) mosaic	
					No.	Type
I						
June 28, 1932	Cumberland	In commercial field; fair vigor; uncaged.[b]	Inoculated	4	4	4D[c]
			Check	5	1	
II						
June 28, 1932	Cumberland	In commercial field; good vigor; uncaged.	Inoculated	6	5	2C; 3D
			Check	5	0	
III						
June 14, 1933	Cumberland	In experimental plot; exceptional vigor; caged; pruned.	Inoculated	5	5	5D
			Check	5	0	
IV						
June 14, 1933	Plum Farmer	In experimental plot; exceptional vigor; caged; pruned.	Inoculated	5	4	4E
			Check	5	0	
V						
June 14, 1933	Cumberland	In experimental plot; extra vigor; uncaged; unpruned.	Inoculated	6	5	1A; 3B; 1C
			Check	6	0	

[a] Aphids were taken at random from the three most vigorous Columbian plantings available and where all plants showed only mild mottling. Aphids for Series I came from one field; for Series II from a second; for Series III, IV, and V from a third.

[b] Cages of cheesecloth were placed over plants in Series III and IV to prevent accidental infections.

[c] As to degree of symptoms exhibited in a period two to sixteen months after inoculation:

A = mild mottling only.

B = medium mottling only.

C = medium mottling; slight necrotic effects on stem tips.

D = medium to severe mottling; decided necrosis on stem tips and leaf petioles.

E = severe or "blistered" mottling; severe necrosis and stunting.

Collections of *Amphorophora rubi*, the most effective aphid vector of raspberry mosaics, were made at random in 3 separate commercial plantings of Columbian raspberries. These source plantings were selected for excellent vigor and uniform mild foliage mottling. From the collections 6 to 20 aphids of varying ages were placed on each healthy test plant. Black-raspberry plants were chosen for inoculation because mosaic symptoms are most clearly and diagnostically expressed on them. Two standard black-raspberry varieties, Cumberland and Plum Farmer, were used. The former is lower in klendusity but higher in resistance to mosaic infection than is the latter.

In total, 23 of 26, or 88.5 per cent, of the black-raspberry plants exposed experimentally to infection by the characteristic "mild mosaic" of the Columbian purple raspberry became infected with red (raspberry) mosaic, according to the Rankin concept of that disease (10, 11). Only 1 of the 26 check plants became infected with red (raspberry) mosaic. This single accidental infection was not unexpected, as both inoculated and check plants in Series I, where it occurred, were exposed to and in no way protected from the natural spread of viruses.

To obtain confirmatory evidence, the order of the above experiments was reversed in 1934. Aphids (*Amphorophora rubi*) were reared under a cheesecloth cage on a Plum Farmer black-raspberry plant that was severely affected with typical red (raspberry) mosaic, the virus having come originally from an infected Ontario red-raspberry plant. Aphids of all ages were taken from this source and 10 were placed on each of 7 vigorous but mildly mottled Columbian plants growing in a commercial field. At the end of 2 months none of the Columbian plants thus exposed to infection exhibited symptoms different from the adjacent check plants. Presumably, then, the inoculated plants already contained the red (raspberry)-mosaic principle and further injection of it produced no apparent consequences.

These experimental results would seem to demonstrate conclusively that the "mild mosaic" mottling in Columbian purple raspberries is actually an expression of infection by a contagious mosaic virus and that this virus is the same as the red (raspberry)-mosaic virus common in black raspberries.

That such is the case has been verified by numerous field observations of natural spread. One decisive example of natural spread of red (raspberry) mosaic from Columbian raspberries into healthy black raspberries is given in the map in fig. 1. In this instance, in the spring of 1931, a 500-lot of virus-free Shuttleworth black-raspberry plants and a similar lot of "mild mosaic" Columbian purple-raspberry plants were set in adjacent plantings. No other sources of mosaic inoculum were present in the

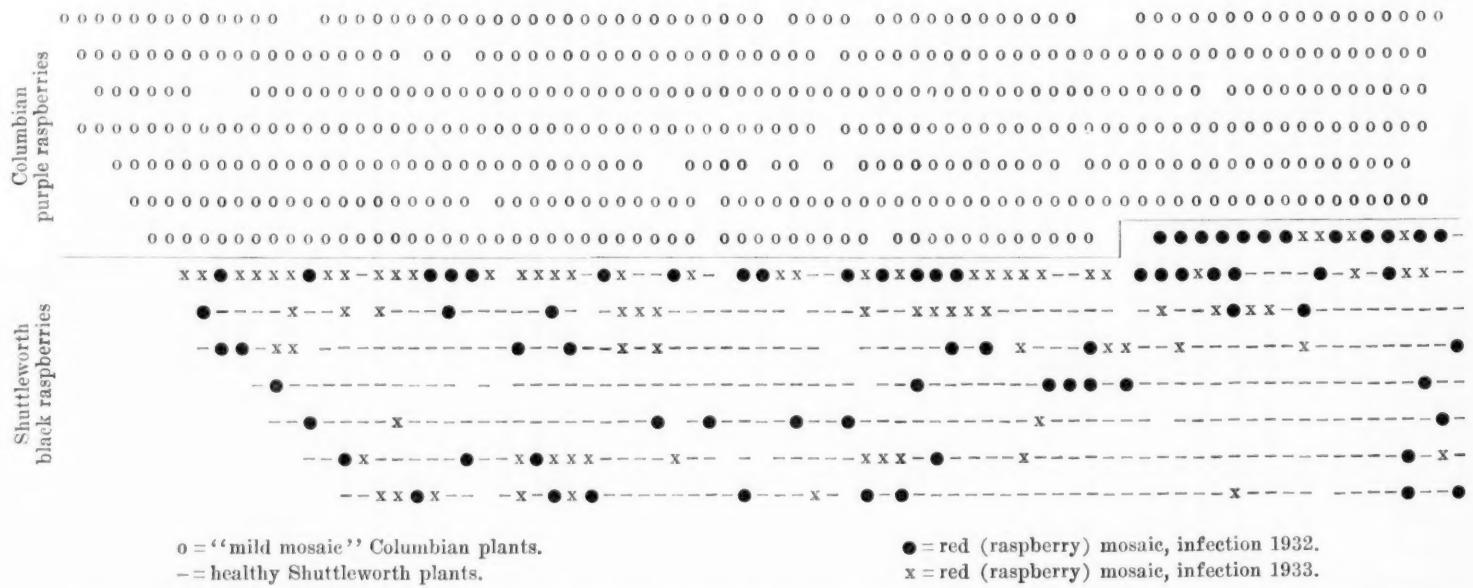
Columbian purple raspberries
Shuttleworth black raspberries
o = "mild mosaic" Columbian plants.
– = healthy Shuttleworth plants.
● = red (raspberry) mosaic, infection 1932.
x = red (raspberry) mosaic, infection 1933.

FIG. 1. Map showing spread of red (raspberry) mosaic into a planting of originally virus-free Shuttleworth black raspberries from an adjacent planting of Columbian purple raspberries uniformly affected with "mild mosaic" only. Both varieties planted in May, 1931; records taken June 25, 1932, and July 13, 1933. Farm of Borthwick and Hall, Silver Creek, New York.

vicinity. June 25, 1932, 73, or 15 per cent, of the 487 living Shuttleworth plants had contracted red (raspberry) mosaic according to the Rankin concept. These were rogued from the field by the growers. One year later, 81, or 20 per cent, of the remaining 414 black-raspberry plants were affected. Of 17 plants in the short row nearest the Columbians, 16 became infected during the two seasons of exposure. In the first long row adjacent to the Columbians, 54 of 71 plants contracted the disease. The distribution of this spread and the rate at which it occurred demonstrated that the "mild mosaic" Columbian plants were the source of the red (raspberry)-mosaic virus. Fields of the variety in general would seem to represent serious menaces to any near-by plantings of virus-free raspberry stock.

Varying Reaction of Black Raspberries to the Columbian "Mild Mosaic" Virus. A development of broader significance, however, was noted in this field experience. Here, to judge by the uniformity of mild symptoms displayed on the host Columbian plants, there could have been present but one common source of but one mosaic virus. Yet all degrees and phases of raspberry mosaic, other than yellow mosaic, were represented in the black-raspberry plants that became affected!

By referring to the last column in table 1 it will be seen that a similar phenomenon occurred in the black-raspberry plants experimentally inoculated with "mild mosaic" from Columbian plants. Of the 23 plants in these tests that developed infection, 1 extremely mild, 3 mild, 3 medium, 12 severe, and 4 extremely severe cases of red (raspberry) mosaic were observed.

The 4 Plum Farmer plants experimentally infected in Series IV were injured most severely of all. In field observations this variety has been noted as susceptible in the extreme to the red (raspberry)-mosaic virus.

The more resistant Cumberland plants used in the other 4 series varied in reaction. This variability seems traceable to growth features. In Series I subject plants were growing in a sandy soil. Their vigor had been average but, at about the time of inoculation, a prolonged summer drought and too deep cultivation with consequent root pruning brought their growth to a sudden stop. The type of infection obtained was uniformly severe for the variety. In Series II subject plants were in originally better vigor than those in Series I and, although affected by drought, had superior culture. Apparently, as a consequence, injury from infections here was somewhat less severe and less uniform than in Series I.

In Series III inoculated plants were on extra fertile soil and grew with extraordinary vigor and rapidity throughout the spring and early summer. Cheesecloth cages were placed over them in early June prior to inoculation and left until October. The shading of cages is conducive to abnormally

rapid growth and succulence in raspberry plants. Three weeks after inoculations in this series extremely mild mottling was evident on 2 leaves near the tip of only one lateral on one plant. At this time it was necessary to prune the plants in order to keep them within the confines of the cages. When the sublaterals produced from axillary buds subsequently had grown to a few inches in length, all of them on all the plants were necrotic on their tips and the mottling of their leaves was pronounced. The pruning, of course, had brought an abrupt check in the rank growth. It is thought that this allowed the virus time to reach and multiply in the growing points of the subject plants in uniform concentration and converted what would probably have been extremely mild (A degree) cases into severe (D degree) cases. The same pruning was done in Series IV and probably was responsible for additional and uniform severity of infection there on the more susceptible Plum Farmer plants.

In Series V the plants used were of the Cumberland variety, were growing in the same row, and, except for caging, were under conditions identical with those in Series III. Their growth, though not so rapid as in Series III, was unusually and steadily vigorous all summer. The pruning of laterals was not practiced on them. Here, one plant showed extremely mild mottling on one lateral in September and the next June it was a medium (C) case. Not until the spring following inoculation did the other 4 plants infected show symptoms and these were extremely mild (A) and mild (B) in degree of severity.

Since the inoculum was obtained in all cases where Columbian plants were affected uniformly with the mild mottling and where no other mosaic symptoms were present, it would seem improbable that more than one virus was concerned or that there could have been present several strains of one virus varying in virulence. The most probable conclusion has seemed to be that only one virus was involved and that the varying reactions obtained were due to varietal differences and to the influence of environmental conditions as they affected growth of each individual inoculated black raspberry plant.

CONCEPTS OF RASPBERRY MOSAICS

To understand the application of the above evidence on the identity of the "mild mosaic" in Columbian hybrid raspberries to the broader question of the identities of raspberry mosaics in general, it will be necessary to develop the entire issue by a chronological review of literature.

Review of Literature

From studies made in Ontario, Canada, on red raspberries, Rankin, Hockey, and McCurry (12), in 1921, presented symptom studies and field experiences that first separated mosaic as a virus-disease entity distinct

from other raspberry maladies. Wilcox and Smith (14), working in northern Ohio, in 1924 reported the first experimental inter-specific transmission of a mosaic virus, from red to black raspberries. Inoculation of this virus into black-raspberry plants produced wilting, stunting, and necrosis of tips with rather severe mottling. For the next several years, this reaction of black raspberries was adopted by investigators as the type of what came to be called "red-raspberry mosaic." In 1926, Dodge and Wilcox (7) described 1 type of mosaic on red raspberries and 3 types on black raspberries without naming them specifically. In March, 1927, Rankin (9), while commenting on varying symptom types in black raspberries, still discussed investigations with mosaic of raspberries in New York from the general standpoint of one disease.

Bennett (1), in Michigan, in May, 1927, used a classification of 3 mosaic types applied to all 3 species of raspberry, and called the types mild mosaic, red-raspberry mosaic, and yellow mosaic. Two years later, he (3) suggested that perhaps "a larger number of viruses than had heretofore been recognized" were responsible for the mosaic disease complex of raspberries. This suggestion was based on a great diversity in symptom expressions obtained in experimental transmission tests. Yellow mosaic was regarded as a distinct type, with mild and red-raspberry mosaics as intergrading extremes of another general type. Rankin (10), in 1930, published an account of field studies in New York in which a correlation of symptoms indicated that mild and red (raspberry) mosaics in black raspberries were caused by the same virus. In the same year, from observations in Michigan and Ohio, Bennett (4) described 4 types of raspberry mosaic, *viz.*, mild, medium, severe, and yellow, but stated that the number of viruses involved had not been determined. In a bulletin on virus diseases of black raspberries in 1931, Rankin (11) made a formal classification of raspberry viruses in which 3 mosaic viruses were listed, *viz.*, red (raspberry) mosaic, yellow mosaic, and mild mosaic. He expressed doubt, however, as to the existence of a distinct virosis that should be called mild mosaic.

In 1932, Bennett (5) published results of experimental transmission tests made in Michigan with 3 selected types of raspberry mosaics other than yellow. These were classified as mild red-raspberry mosaic, medium red-raspberry mosaic, and severe red-raspberry mosaic. Each type was reported to have remained stable in degree of symptom expression through series of transmissions on red (variety King) and black (variety Cumberland) raspberries during a period of 3 years. The possibility of several strains of one virus with varying virulence was suggested to account for the results obtained.

In a general treatise on raspberry diseases, Rankin (5), in 1933, without presenting further evidence or discussion, followed a two-virus classification, namely red (raspberry) mosaic and yellow mosaic.

Some salient points are worth comment. All investigators are agreed on the individuality of the yellow-mosaic virus. The controversy, then, resolves itself into whether one or more than one virus is involved in the production of the wide range of mosaic symptoms on raspberries known not to be due to the yellow-mosaic virus. Argument is possible over this question because the said range of symptom extremes extends from a very light mottling visible only early in the season and accompanied by no appreciable injury *through all gradations* to a "blistered" mottling associated with severe necrotic, growth-stunting, and rapid-killing effects. Complicating factors, such as weather, plant vigor, three species of hosts with numerous varieties, and possible association with the yellow-mosaic virus, all affect symptom expression and thus increase the chances for confusion and contradictory opinions as to causal relationships. One investigator may explain satisfactorily that a single virus causes all the varied symptoms and that their diversity is due to varietal and individual plant susceptibility or reaction, as modified by weather, soil, and growth factors. Another may contend with equal plausibility that, since the diversity of reaction is often great within even a single planting of one variety, the different types of disease could be produced only by different viruses or combinations of viruses, or at least by distinct strains of the same virus differing in virulence.

One highly significant point is that, although the range of severity of symptoms is wide, it is *graduated.* Bennett (3, 5) has twice mentioned the lack of clear demarcations between his selected types of mild, medium, and severe red-raspberry mosaics. Apparently 4, 5, or a dozen representative types of disease severity may be selected as well as 3. The number of divisions to be made would seem to depend on the investigator's desire for detail rather than on the existence of so many distinct types in nature.

Another interesting feature is the evolution that the application of the term red-raspberry mosaic, or its other forms, red (raspberry) mosaic and red mosaic, has undergone from an originally restricted scope to its present broad usage. Regardless of their opinions as to the number of viruses involved, all investigators are now using this name to include all types of mosaic in raspberries other than yellow mosaic.

Application of the Columbian "Mild Mosaic" Studies

The present experiences with "mild mosaic" in Columbian hybrid raspberries appear to have demonstrated not only that this mosaic is the red (raspberry) mosaic, but that one strain of this one virus may produce the entire range of other-than-yellow mosaic symptoms that have been observed on black raspberries in eastern North America. These findings support the Rankin theory (10, 13) that there are but two known rasp-

berry mosaic viruses in this region, namely, red (raspberry) mosaic and yellow mosaic. When put to test in extensive field work, this concept has proved satisfactory and fitting in every situation.

Final and indisputable decision as to the number of raspberry-mosaic viruses cannot be made, of course, until the viruses can be isolated and studied *in vitro*. But there is no immediate prospect of this being possible as they seem to perish almost simultaneously upon extraction. As long as this situation exists, the opinions of investigators, even though drawn from experimental inoculation trials, will be based necessarily upon their observations of symptoms and interpretations thereof. And such opinions, of course, will be open always to question.

However, practical dealings with raspberry mosaics require the formation of definite concepts. For the present, then, the general adoption of the 2-virus classification is suggested, as it not only appears to be the more accurate but possesses the additional advantages of being simpler and clearer.

PROPOSED CHANGE IN TERMINOLOGY

Although contradictory explanations still may remain as to why the differences in degree of symptom severity exist in red (raspberry)-mosaic infections, as pointed out in the literature review above. American investigators are now using essentially the same name for the disease. But the term "red-raspberry mosaic" and all its derived forms seem objectionable in several respects, especially to those persons not intensively acquainted with raspberry virus diseases.

"Red-raspberry mosaic" is not only a cumbersome name, but it is misleading. This particular mosaic is not limited to nor is it of most common occurrence in red raspberries; in fact, it is more frequent and more distinct in black and purple than in red raspberries. The term "red-raspberry mosaic" was derived originally from the interspecific transmission of the virus from red to black raspberries reported by Wilcox and Smith (14) in 1924. "Red mosaic" was introduced into literature by Rankin (10) in 1930 simply as an abbreviation of its predecessor. This name carries the same likelihood of misconstruction, *i.e.*, that the disease occurs principally in the red-raspberry species. In addition, it is mistaken frequently as having reference to a diagnostic color feature in the disease symptoms. Such misunderstanding is apt to occur particularly because "red mosaic" seems contradistinctive to the name "yellow mosaic" that is descriptive of the disease in raspberries to which it is applied.

The discarded terms "mild mosaic," "medium mosaic," and "severe mosaic" (4) have been used as descriptive of degrees of what now appears to be one disease. No one of them alone satisfactorily denotes the entire complex of the disease.

There seems to be no reason to continue the use of any of these names in view of their inappropriate connotations. *Green mottle mosaic* is suggested here as the most logical and suitable substitution for them. This name is descriptive of the fundamental symptom of the disease, all species and varieties of raspberries considered. With this disease, the severity of mottling is known to vary extremely but, whenever shown, consists of a contrast between light green and dark green areas in the leaves. The only known exception to this general rule is to be found occasionally on fruit-spur foliage of seriously injured individual plants of a very susceptible black-raspberry variety like Plum Farmer. In such restricted instances the foliage may be a solid green but of much deeper intensity than normal.

The name "green mottle mosaic" also portrays the general contrast in chlorotic symptoms that distinguishes the 2 raspberry mosaic diseases. A true mottle pattern is characteristic of green mottle mosaic, while the chlorosis, evident in yellow mosaic, is not strictly a mottling but a general yellowing of tissues that affects small portions of leaves or entire leaves. This yellowing is progessive in individual leaves from their veins outward and their margins inward.

The yellow mosaic virus tends to produce more leaf deformity in all species of raspberries, but this difference is not constant. There is another difference between the 2 diseases in the position of leaves on affected plants in which chlorosis appears. Green mottle mosaic is seen most commonly in a host plant on the lower leaves of the spurs on fruiting canes and on the lower leaves of the turions, whereas yellow mosaic is expressed on the upper leaves of the fruit spurs and on the higher leaves of turions. This position difference, however, is relative and intergrading and varies between seasons and varieties according to weather, host growth conditions, and length of time plants have been infected. Neither host position nor leaf-deformity differences are deemed suitable criteria for founding distinctive names for the raspberry mosaic diseases. Besides, renaming on either of these bases would require redesignation of both diseases.

CONCLUSION

In brief, it is believed that the situation in regard to raspberry mosaics would be simplified and clarified for many and would be presented more accurately according to the results of experimentation and observation up to the present time in eastern North America if the following classification of them were adopted.

Raspberry Mosaics:

Virus 1. Green Mottle Mosaic. This name is recommended as a substitute for the terms "red-raspberry mosaic" and "red mosaic." It is

meant to include the entire range of symptom expression from very mild to very severe cases; in fact, all types of mosaic of raspberry other than the distinct cases known to be due to the yellow-mosaic virus. For convenience, the term "green mottle mosaic" might well be contracted to "green mosaic."

Virus 2. Yellow Mosaic. All investigators are agreed upon the usage of this name. No change is needed or advised.

SUMMARY

Experimental transmission trials and field studies in disease spread have proved that the "mild mosaic," universally present in Columbian hybrid raspberries, is caused by a virus identical with the red (raspberry)-mosaic virus.

In these tests, the inoculated black-raspberry plants developed all degrees of other-than-yellow mosaic symptoms from extremely mild to extremely severe phases. Since the mosaic inoculum was obtained from sources where its presence had been uniform in expression, the results were interpreted as demonstrating that a single virus may produce all the mosaic types other than yellow mosaic that occur in black raspberries in eastern North America. This indication has been substantiated by field experiences.

Knowledge of the environmental, climatic, and cultural conditions that affected the black raspberry plants inoculated in these experiments indicated that such factors, through their effect on host growth, influenced the severity of reaction obtained in individual cases.

These findings support the Rankin theory that there are only two viruses involved in raspberry mosaics of eastern North America, namely, red (raspberry)-mosaic and yellow-mosaic.

The name "red-raspberry mosaic" and its derivatives are confusing in connotation. The much more fitting term *green mottle mosaic* is suggested as a substitute. For the sake of brevity, this name might be shortened to "green mosaic."

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THE PARASITISM OF ARCEUTHOBIUM (RAZOUMOWSKYA) CAMPYLOPODUM ON PINUS JEFFREYI

JEAN DUFRÉNOY

(Accepted for publication February 27, 1935)

Histological studies of the relations between the parasitic Loranthaceae and pines date back to 1905, when Peirce[1] studied the germination of *Arceuthobium* (*Razoumowskya*) *occidentale* on *Pinus radiata.* He showed that when the root tip of the germinating embryo comes in contact with a slight elevation of the surface or the base of a bundle of needles, it forms a holdfast consisting of a mass of parenchymatous cells, which penetrates, haustorium-like, and branches into the medullary rays of the host.

Vascular tissues are formed at an early stage, and Peirce has shown the "direct contact of a tracheid of the parasite with a tracheid of the host;—in this perfect connexion of the xylem-tissues of parasite and host we have nothing uncommon."

The writer found the same relationship (Fig. 1) between tracheids of *Arceuthobium* (*Razoumowskya*) *campylopodum* and the tracheids of *Pinus jeffreyi,* in material made available through the courtesy of Dr. E. P. Meinecke. The haustorium of the parasite, pushing into the medullary ray of the host, causes hyperplasia of the cells of the medullary ray, and inhibits the normal differentiation of the xylem elements in the immediate vicinity. Instead of perfectly parallel elongated tracheids showing the normal bordered pits, elements are formed with irregular woody thickenings of their walls.

Figure 1 shows, in a radial section of an infected twig of *Pinus jeffreyi,* the xylem of Arceuthobium (R) descending from the upper parts of the parasite through a medullary ray (M) of the pine, there to end in close contact with the xylem elements of the pine wood.

Peirce emphasized the relations of the phloem tissues of Arceuthobium with those of pine, but he "sought vainly for anything more than contact between the cells of Arceuthobium and the sieve tubes of pine." Neither did the writer find anything more in Jeffrey pine, nor did he expect it. Arceuthobium, like other such parasites, shows little histological differentiation; besides, it is felt that in the parasitic relationship, as well as in the whole problem of translocation of materials through the vascular plants, the anatomical and histological aspects have been overemphasized to the point of overshadowing the physiological side. What we are concerned

[1] Peirce, George J. The dissemination and germination of *Arceuthobium occidentale* Engl. Ann. Bot. 19: 99–113. Jan. 1905.

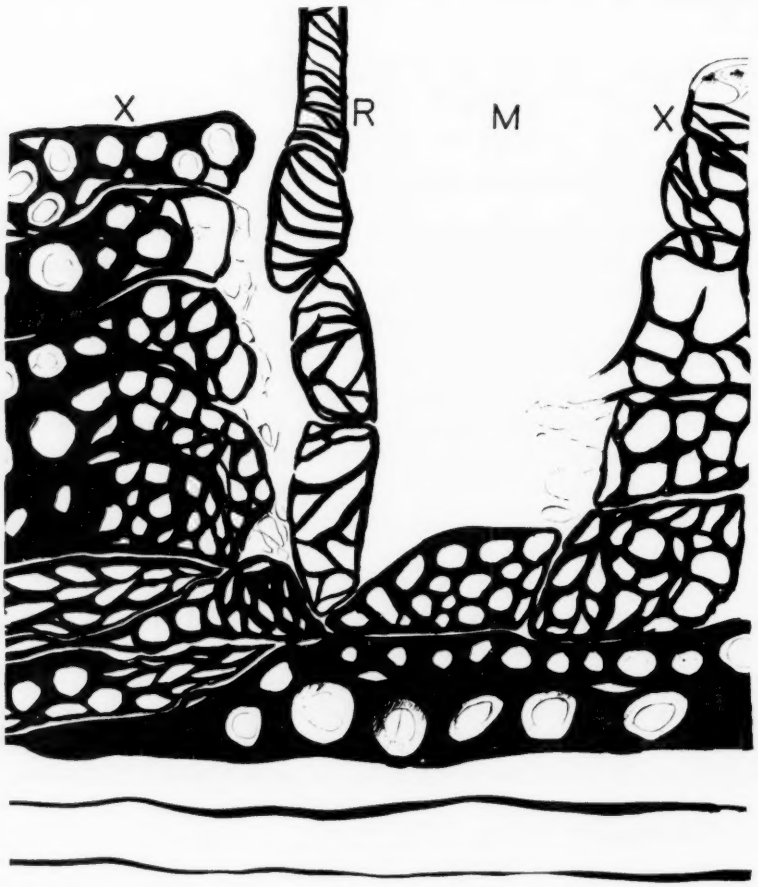

FIG. 1. Section through a twig of *Pinus jeffreyi*, showing the xylem of *Arceuthobium campylopodum*, R, in close contact with the pine wood-elements, X. All lignified tissues, staining red with safranin, are shown in solid black. Bordered pits are outlined. M, nonlignified portions of wood.

with is primarily a problem of relation between the two living organisms and, therefore, a problem of relationship between the living cells of both.

The problem should be studied in sections of living material. Free-hand sections, stained with a solution of neutral red, prepared in the Ringer fluid, plainly show the importance of living parenchymatous cells where the parasite comes into contact with the host. The main effect of the parasite is to induce the formation of wide medullary rays, thereby reducing the formation of wood vessels.

The parasite itself is made up of a mass of parenchymatous cells, with merely an axial row of elongated cells, showing lignified thickenings

of the cell walls and representing rudimentary wood vessels. Elongated living cells, surrounding these wood vessels, may be interpreted to represent the phloem tissues. Each living parenchymatous cell, either of the parasite or of the host, shows one or a few vacuoles, containing a watery solution that stains red with neutral red. Where the haustorium comes into contact with the tissues of the pine, each cell shows a number of small vacuoles. (Figs. 2 and 3.) The presence of many small vacuoles in a cell is correlated with an active translocation of soluble materials, and generally with that of polypeptides.

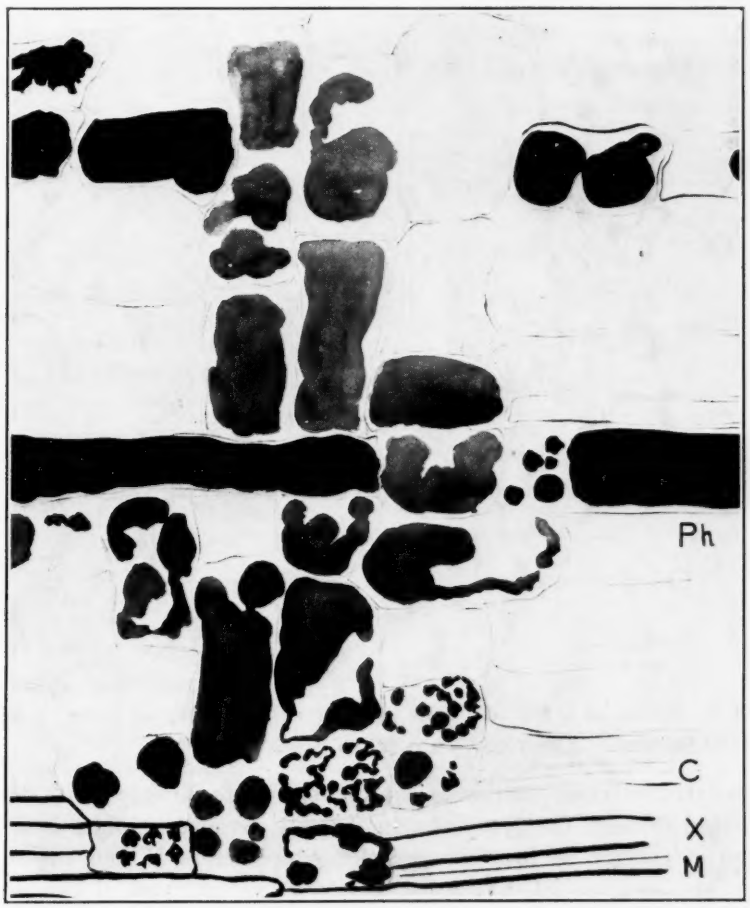

FIG. 2. Section of twig of *Pinus jeffreyi*, showing phloem Ph, cambium C, xylem X, and medullary ray M, penetrated by the cells of Arceuthobium. Small vacuoles, the contents of which are stained red by neutral red in the living cells, are shown in black; vacuoles staining pink are shown in grey.

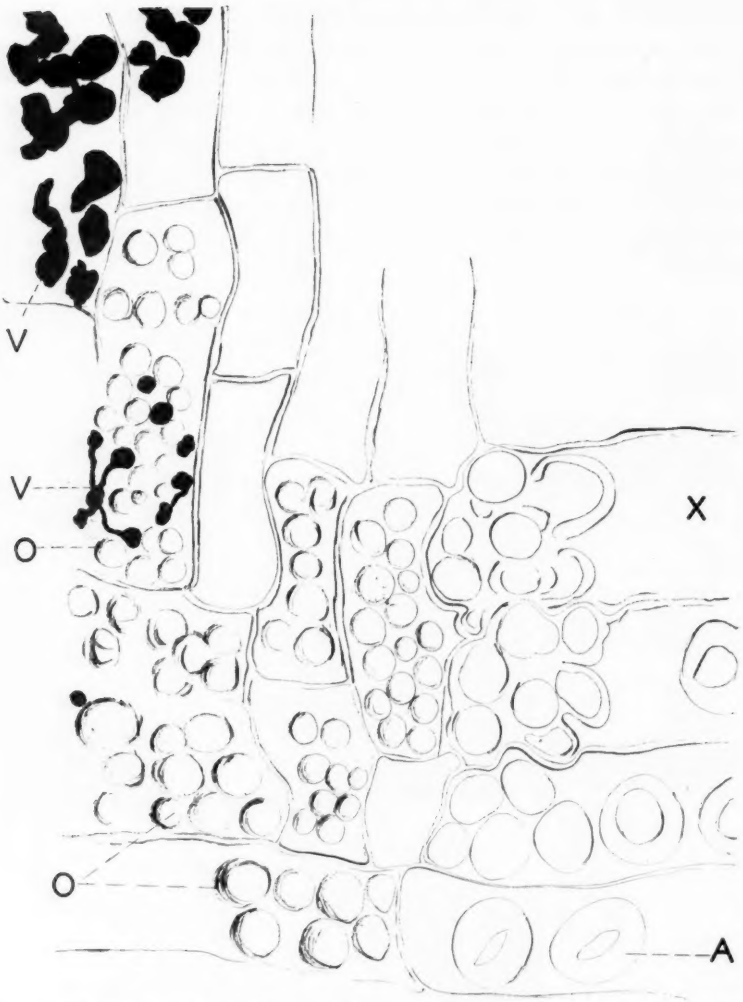

FIG. 3. Details of a few cells from the same medullary ray as shown in figure 2. V indicates vacuoles, O oleoresin drops, X xylem, A bordered pits.

The extreme fragmentation of the vacuolar material into many droplets distributed through the cytoplasm in the cell obviously results in a great increase of contact surface between the cytoplasm itself and the surface of the vacuolar material. From the results of recent cytological investigations it appears that most of the activities of the living cell, particularly the respiration process, are mainly located at those surfaces of contact between the cytoplasm and the vacuoles, so that the greater the surface of contact, the more active the cell.

We may, therefore, infer from the cytological aspects of the living cells that, where a haustorium of Arceuthobium comes in contact with the medullary rays of the pine, an active translocation of nutrients takes place, presumably as the result of enzymatic activity by the cells of the haustorium.

The cells of the infected medullary rays are rich in droplets of oleoresins. The tissues of the Arceuthobium, imbedded in the cortex of the pine branch, as examined in the month of February, show an abundance of starch. This may, in part, explain why porcupines are especially fond of feeding on them.

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A PROTECTIVE ZONE IN RED GUM FIRE SCARS

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(Accepted for publication March 25, 1935)

INTRODUCTION

Of several southern hardwood species studied in a recent pathological investigation in the Mississippi Delta, red gum, *Liquidambar styraciflua*, and persimmon, *Diospyros virginiana*, appeared to be the most resistant to decay infection through fire scars (Table 1). Shortly after being scarred

TABLE 1.—*Percentage of fire-scarred trees decayed in several southern hardwoods*[a]

Species	Number of trees examined	Percentage of trees decayed
Hackberry (*Celtis laevigata*)	23	100
Nuttall Oak (*Quercus nuttallii*)	33	82
Overcup Oak (*Q. lyrata*)	10	80
Water Oak (*Q. nigra*)	32	72
Ash (*Fraxinus* spp.)	19	47
Red Gum (*Liquidambar styraciflua*)	85	42
Persimmon (*Diospyros virginiana*)	5	20

[a] A tree was considered decayed if rot had progressed ½ in. or more in from scar surface. As a rule a tree designated as decayed was definitely decayed, as in figure 1, B. All trees were scarred 8 years before dissection and at no other time before or after that date.

by fire both of these species form hard dark zones on the surfaces of the scars, extending to a depth of about 2–10 mm. into the wood. Other species, particularly ash, *Fraxinus* spp., also tend to form such zones but the zones are not so well developed as in red gum and persimmon. These zones appear to offer a protection from decay to the wood beneath. The protective zone in red gum was studied in detail.

MACRO- AND MICROSCOPIC APPEARANCE OF PROTECTIVE ZONE

Figure 1, A, shows a section through a young red gum scarred by fire 22 years and again 15 years before dissection. The dark protective zone is plainly visible around the edges of the scars. Even though the scars on this tree exposed a considerable portion of the sapwood at the base, the tree remained sound. In a manuscript[1] by the senior author shortly to be published, it is shown that red gum is more subject to decay from fire scars if

[1] Unpublished manuscript "Decay Following Fire in Young Mississippi Delta Hardwoods" by George H. Hepting.

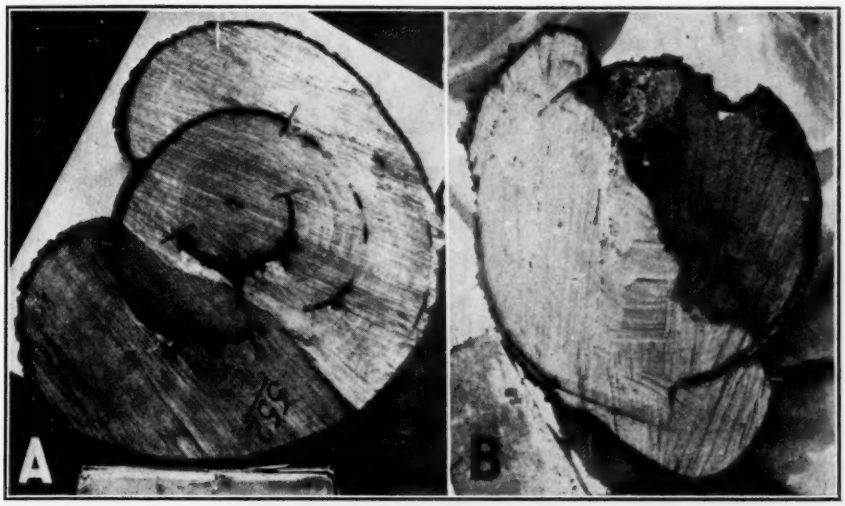

FIG. 1. A. Cross section through base of a young red gum scarred by fire 22 years, and again 15 years before dissection. × ⅓. Note protective zones and absence of decay behind scar. B. Cross section through base of young red gum scarred on one side 4 years and on the other side 2 years before dissection. × ⅓. Note disruption of protective zone on one side, and decay of sapwood behind scar.

burned more than once than if burned only once. Figure 1, B, shows a section through a young red gum scarred on one side 4 years and on the other side 2 years before dissection. In spite of the relatively short time since this tree had been scarred, there was considerable decay of the sapwood behind the older scar. Note that while the protective zone is plainly visible it has been broken at one place.

The protective zone in red gum fire scars usually comprises several of the outer layers of sapwood cells, although it may be formed some distance in from the surface. The zone appears similar to normal heartwood except that it is darker, much harder, and is heavily infiltrated with a brown substance that appears to be a gum and will be referred to as gum in this article.

Sections were prepared of the protective zone, and these were examined under the microscope. There was about one millimeter of sapwood on the face of the block in which no gum was deposited. The gum zone was behind these cells. This was about 2 millimeters in extent in the sections examined. Behind this zone was sapwood in which no gum was found in the wood fibers and tracheids. Gum had, however, been produced in the medullary-ray cells and in some of the wood-parenchyma cells for a considerable distance from the zone proper. The cells within the zone were heavily impregnated with a dark brown gum (Fig. 2, A, and C). The ray and wood-

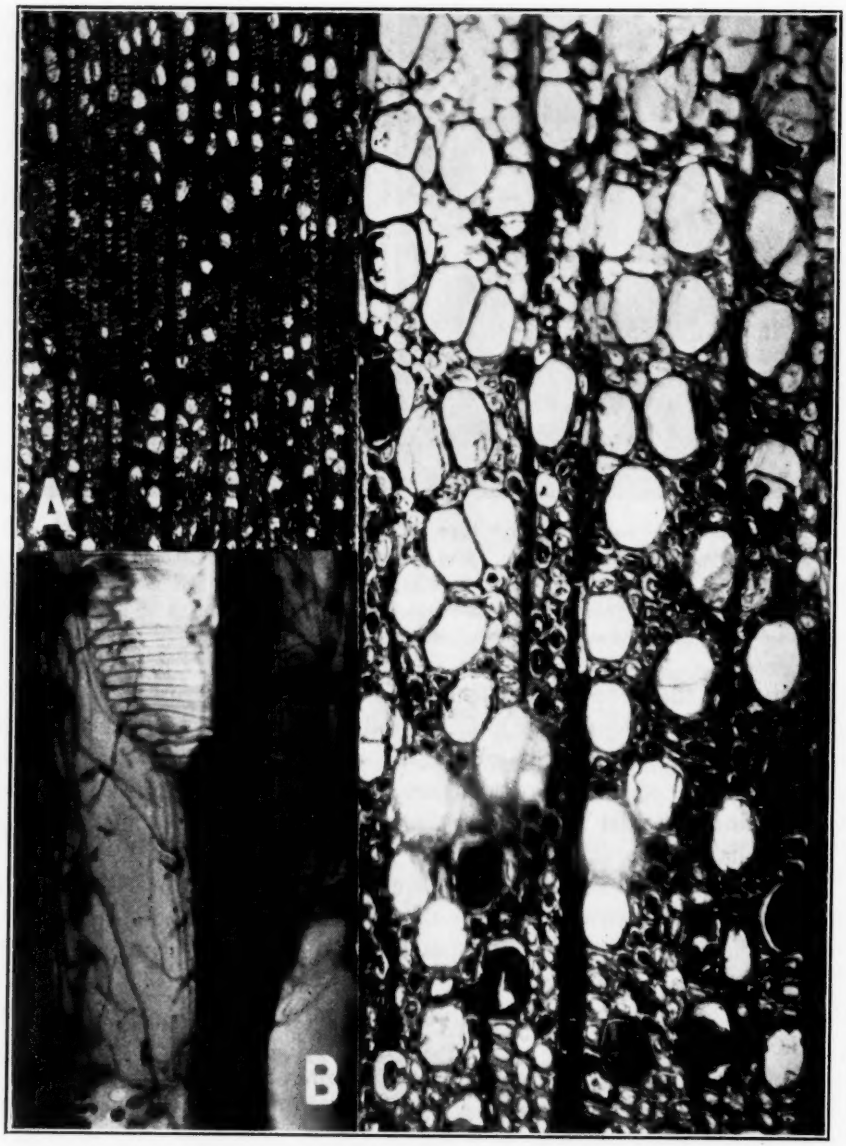

FIG. 2. A. Cross section taken at outer edge of red gum fire scar, showing protective gum zone formed just back of scar edge and normal wood beyond gum zone. The scar edge is at bottom of A. B. Mycelium of *Polyporus pargamenus* in vessel and parenchyma cell within gum zone. C. Cross section taken at border of gum zone (lower part) and originally normal sapwood (upper part) after block had been subjected to decay by *P. pargamenus* for one year. Note decomposition of cell walls in originally normal wood and their preservation within gum zone.

parenchyma cells were completely filled with gum in the densest part of the zone and the lumina of the fibers were nearly filled. Even the largest vessels usually contained considerable gum, this being found as a hard brown covering or thick film over the inner cell walls in some and in others in the form of globules. (Fig. 2, B, lower end of vessel.)

RESISTANCE OF PROTECTIVE ZONE TO DECAY IN ARTIFICIALLY INOCULATED BLOCKS

Several small blocks about $\frac{1}{4}$ in. square and $\frac{1}{2}$ in. long were cut from the outer part of a red gum fire scar. The blocks included part of the protective zone and about $\frac{1}{4}$ in. of sapwood adjacent to and behind it. These blocks were placed in test tubes with water and autoclaved for $\frac{1}{2}$ hr. at 15 lbs. pressure. *Polyporus pargamenus* Fr., growing on agar, was placed on some and on others *P. gilvus* (Schw.) Fr. was planted. These 2 fungi were used, since they are well-known rotters of dead sapwood. The tubes were then allowed to stand for a year at room temperature. At the end of that time the blocks were examined. A dense fungous mat completely covered each of the blocks. Upon removal of the mat it was found that the previously normal sapwood was completely decayed both by *P. pargamenus* and *P. gilvus* so that it crumbled under slight pressure between the fingers. The adjacent gum-filled zone, however, appeared to be as hard and firm as it had been prior to inoculation.

The decayed blocks were placed in an alcohol-formalin-acetic fixing solution. After remaining in this solution for 48 hours or longer the blocks were dehydrated by passing them through a series of butyl alcohol and infiltrated with paraffin.[2] The blocks were then embedded in paraffin and the paraffin blocks were soaked in water to facilitate cutting. Sections were cut on a rotary microtome. Some of the sections were stained with Flemming's triple stain and others with the methyl violet-Bismark brown stain.[3] The latter stain was found to be the most effective. Sections of blocks inoculated with *Polyporus pargamenus* or *P. gilvus* were compared with each other and with those from noninoculated blocks. Both fungi completely decayed the sapwood adjacent to the protective zone.

Upon examination of the sections from blocks that had been subjected for a year to decay by each of the two fungi it was found that neither the position nor extent of the protective zone had changed. There seemed to be a little less gum in the cells in some of the sections of decayed wood but, since this was not so in all of them, the loss of gum may have been due to the

[2] Zirkle, Conway. The use of n-butyl alcohol in dehydrating woody tissue for paraffin embedding. Science n.s., **71**: 103–104. 1930.

[3] Hubert, E. E. A staining method for hyphae of wood-inhabiting fungi. Phytopath. **12**: 440–441. 1922.

embedding or staining processes. The cells in the gum region showed no signs of decay and appeared to be in as good condition as in undecayed wood. The cells beyond the gum area, however, were very much decayed (Fig. 2, C). In most cases the tertiary and secondary thickenings of the cell walls had almost, if not entirely, disappeared, only the middle lamellae of the cells being left. In some places the middle lamellae had become so thin and brittle that the connections between cells had become entirely obliterated, only fragments remaining. The ray cells and the wood-parenchyma cells, which had been filled with gum, were undecayed.

Considering the excellent conditions for decay and the relatively long time these small blocks were submitted to fungous action the gum-filled zone showed great resistance to the action of the two fungi used, in contrast to the readiness with which the normal wood decayed.

The fact that the cells in the gum-filled zone did not decay in the laboratory tests is not entirely explainable by any failure of the mycelium to penetrate this zone, because, as shown in figure 2, B, mycelium was abundantly present in many of the wood elements in this gum zone that were not completely filled with gum. The zone appears to be similar to heartwood that has been heavily infiltrated with gum. The resistance of this zone to decay by the fungi may thus be similar to the resistance of normal heartwood to the action of sapwood-rotting fungi, with the added mechanical resistance of the heavy infiltration of hardened gum. In the present tests the gum appeared not to be utilized by the fungi, as it was still abundant at the end of the incubation period of a year. In no case were hyphae found penetrating a gum globule or passing through a completely gum-filled cell. It is probable that, under natural conditions, fungus mycelium could penetrate all the way through the gum-filled zone only with great difficulty, if at all. Under the laboratory tests the fungi were permitted to become well established around the gum area and could readily enter the vessels in this area through their open ends. Under natural conditions the proximity of the zone to the outside surface of the scarred area would leave a fungus little normal wood in which to become established. Penetration to the underlying sapwood also would have to be in a radial direction through dozens of gum-filled cells.

The protective zone formed in a red gum fire scar, through its impervious nature resulting from heavy gum infiltration, apparently acts to preserve the living sapwood beneath the surface of the scars from drying and subsequent decay. Through its resistance to decay this zone may remain an effective seal for many years. Many cases have been observed where large red gum fire scars (Fig. 1, A) have completely healed without any decay development behind the scarred area. If a once-scarred red gum is exposed to more fires the sapwood beneath the protective zone may be killed

by heat, and decay may then readily take place (Fig. 1, B). The reason that the sapwood behind the protective zone decays more readily when killed may be because subsequent checking allows ingress of fungi behind the zone. It also may be that wood-destroying fungi are in contact with the wood beneath the zone while the zone is still intact, but are capable of decaying it only if the wood be dead. In some cases the sapwood beneath the protective zone may become exposed to decay through imperfect formation of the zone or through the action of insects, particularly ants and termites, which commonly infest scarred trees in the Delta Region. This zone also may act as a mechanical barrier preventing mycelium from reaching the sapwood beneath.

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PRESENT GENERIC STATUS OF THE CITRUS-SCAB ORGANISM

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Sphaceloma fawcettii Jenkins, the fungus that causes scab of citrus (*Citrus*), is undoubtedly to be classified in the genus Sphaceloma where it was placed (6) in January, 1925. It is the writer's understanding that this is now also the opinion of Doidge and Butler, although in September, 1924, they referred (4) the fungus to the genus Sporotrichum as closely related to the animal pathogens of this "heterogenous" group.

The genus Sporotrichum as amended by Saccardo (9) included two species, *S. virescens* Link and *S. roseum* Link. The former species is excluded by Lindau (8). *S. roseum* may, therefore, be regarded as the type, as is designated by Clements and Shear (3, p. 389), and *Sphaceloma fawcettii* is clearly not related to this species.

A recent parallel cultural comparison of *Sphaceloma fawcettii* with a culture of *Sporotrichum schenckii* (Hektoen and Perkins (5)) De Beurmann and Gougerot (1), the first animal pathogen to have been classified in the genus Sporotrichum (1, 10) suffices to demonstrate that the two are strikingly unlike, as shown below. The culture of the Sphaceloma employed was isolated from scab lesions on sour orange (*Citrus aurantium* L.) from Florida, on October 20, 1928, that of the *Sporotrichum schenckii* was contributed by Charles Thom, of the Bureau of Plant Industry, who obtained it from Rhoda W. Benham, of the Laboratory for Medical Mycology, College of Physicians and Surgeons, New York, N. Y. The cultures were grown on both Sabouraud's maltose agar and on potato-dextrose agar.

As illustrated in Figure 1, A and C, and D to F, they were distinctly unlike on both media, scarcely showing even a superficial resemblance. It will be noted that the 24-day-old culture of *Sphaceloma fawcettii* on the maltose agar (Fig. 1, A) does not cover the surface of the slant as does the Sporotrichum (Fig. 1, C), of which the growth was limited by the area of the slant. This culture and the similar one of *Sphaceloma ampelinum* de Bary, *i.e., Elsinoe ampelina* (de Bary) Shear, as the fungus is now known in its perfect stage, included in the comparison (Fig. 1, B) on maltose agar were both much brighter colored than the Sporotrichum; that of *Sphaceloma fawcettii* was Hay's maroon,[1] with the lighter portion on one side Prussian red, and that of the Elsinoe, mostly Vandyke brown, or of

[1] Ridgway, R. Color standards and color nomenclature. 43 pp., 53 col. pls. Washington, D. C., 1912.

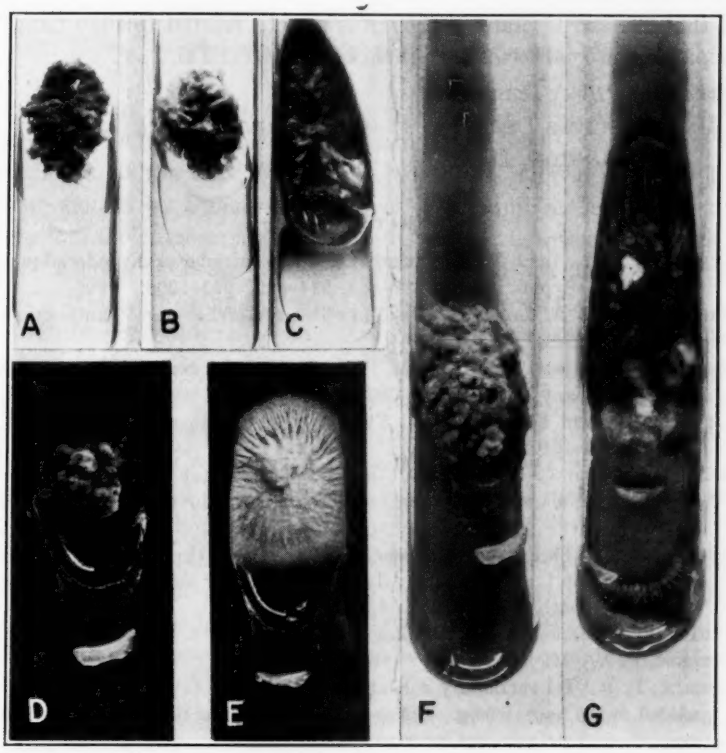

FIG. 1. Parallel cultures of different fungi. A–C. On Sabouraud's maltose agar. A. *Sphaceloma fawcettii*. B. *Elsinoe ampelina*. C. *Sporotrichum schenckii*. D and E. On potato-dextrose agar. D. Sphaceloma. E. Sporotrichum. F and G. Cultures shown in D and E, photographed at a later date. All × 1.

this color mingled with Rood's brown, while the culture of Sporotrichum ranged from drab gray to black.

On potato-dextrose agar the young thallus of *Sphaceloma fawcettii* was raised and irregularly convoluted from the first, and it had scarcely reached the sides of the tube, even after 12 days' growth (Fig. 1, D). On the other hand, the somewhat faster-growing Sporotrichum began its development as an appressed smooth growth with an even margin and with no sign of convolutions for at least 7 days. Numerous radial convolutions developed, however, during the next 5 days (Fig. 1, E). The appearance of these 2 cultures had changed considerably by the time they were 28 days' old (Fig. 1, F and G), or even 2 months' old, but they were still unlike.

In a related paper (7) it is shown that *Elsinoe ampelina* is not pathogenic on rabbits, as previously reported by Charrin and LePlay (2, p. 521–523), and on the basis of the present contribution there is no reason to

believe that the citrus plant pathogen is closely related to such fungi as the human pathogen *Sporotrichum schenckii.*

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PHYTOPATHOLOGICAL NOTE

A Sphaceloma on Fruit of Hesperethusa crenulata, a Remote Citrus Relative from India.—Winston, Bowman, and Bach[1] reported *Clausena lansium* Skeels, of the tribe *Clauseniae,* as moderately affected by Florida citrus scab caused by *Sphaceloma fawcettii* Jenkins, and stated that, with this exception, the rutaceous plants affected by the disease belong to the tribe *Citrinae.* Among these they assert that "members of only 4 genera, viz., Citropsis, Poncirus, Fortunella, and Citrus, are known to be affected by this disease. Only one occurrence on Citropsis and Fortunella was noted and Poncirus was only moderately attacked. These authors explain that Fortunella and Poncirus were only recently removed from the genus Citrus.[2]

Hesperethusa crenulata (Roxb.) Roemer, a remote citrus relative of the tribe *Triphasinae* was not affected by scab in Florida where the work by Winston, Bowman, and Bach, referred to above, was done. But there is a previous record of scab on this host as present on fruits presented to Walter T. Swingle by the Curator of the Royal Botanical Gardens at Sibpur, near Calcutta, British India.[3] These ripe fruits were received in February, 1916, under the Foreign Seed and Plant Introduction number 41947, and were labelled *Limonia acidissima* L. as *H. crenulata* had "commonly but erroneously"[4] been called at that time.

This plant is said to be "native to dry hills in Ceylon, India, Burma, and Indo China."[5] The more or less circular, raised or convex, and sharply delimited scab lesions are shown in figure 1. The causal fungus (*Sphaceloma* sp.) is visible as dark punctiform areas at the center of the lesions on the enlarged fruits (Fig. 1, *B*). The fructifications visible in figure 1, *C*

[1] Winston, J. R., J. J. Bowman, and W. J. Bach. Relative susceptibility of some rutaceous plants to attack by the citrus-scab fungus. Jour. Agr. Res. 30: 1087–1093. 1935.

[2] The botanical classification and nomenclature of the rutaceous plants referred to in this article follow Walter T. Swingle.[a, b, c]

[a] Swingle, W. T. Study of the phytogenetic relationships of the rutaceous sub-family Citrateae, including the citrus fruits and their wild relatives, with experimental studies in the hybridizing and grafting of plants of this sub-family. [In Japanese] Studia Citrologia 1: 1–4 (N. 1, March 1927).

[b] Swingle, W. T. Citrus and related genera. In Bailey, L. H. The Standard Cyclopedia of Horticulture. New York. See V. 1, p. 91 (1914), V. 3, p. 1478, fig. 1825 (1915).

[c] Swingle, W. T. The name of the wood apple, *Feronia Limonia.* Jour. Wash. Acad. Sci. 4: 325–328. 1914.

[3] U. S. Department of Agriculture Federal Horticultural Board Letter of Information No. 22, p. 6. 1916.

[4] *Loc. cit.* See footnote c.

[5] *Loc. cit.* See footnote b.

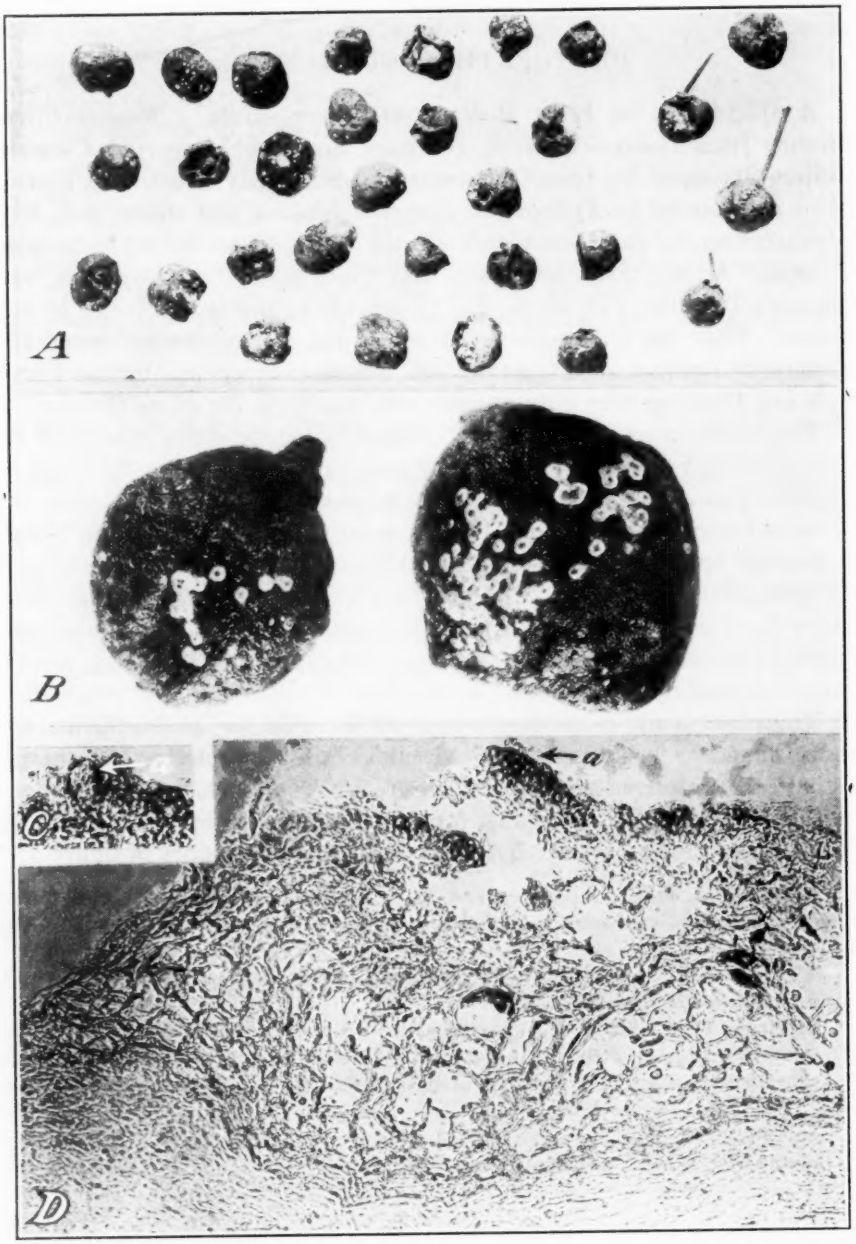

FIG. 1. *A* and *B*. Scabbed fruits of *Hesperethusa crenulata* from British India (*A*, ×1; *B*, ×5½). *C* and *D*. Sections showing (*D*) the hyperplastic character of the lesions and (*C*, *a* and *D*, *a*) fructifications of the *Sphaceloma* as explained in the text (*C*, ×310; *D*, ×275).

and *D*, consist principally of a mass of microconidia (Fig. 1, *C*, *a*) and acervuli (Fig. *D*, *a*).

Further critical study of this Sphaceloma is, of course, essential for its specific or varietal identification. The symptomatological characters have a certain similarity to those of sweet orange fruit scab, known only in South America and attributed to *S. fawcettii viscosa* Jenkins. This similarity is suggested by a comparison of figure 1, *B*, here shown with a closely corresponding illustration of sweet orange fruit scab previously published.[6]

For some time it has been thought advisable to search for Sphaceloma on *Rutaceae*, particularly the more or less remote citrus relatives belonging to the subfamily *Citratae*, in the hope of obtaining more helpful information on the origin of the different forms of citrus scab, such as the sweet orange fruit scab referred to above and Australian citrus scab caused by *S. fawcettii scabiosa* Jenkins. The present study may be a beginning. Both Hesperethusa and Clausena, although rather remote relatives of Citrus that do not belong to the tribe *Citrinae*, are, nevertheless, closely enough related to Citrus to be grafted on it, as shown by Swingle.[7]—ANNA E. JENKINS, Bureau of Plant Industry, U. S. Department of Agriculture, Washington, D. C.

[6] Jenkins, A. E., and H. S. Fawcett. Records of citrus scab family from herbarium specimens of the genus citrus in England and the United States. Phytopath. **23**, p. 476, fig. 1, A and B. 1933.

[7] *Loc. cit.* See footnote c.

REPORT OF THE SUMMER MEETING OF THE AMERICAN PHYTOPATHOLOGICAL SOCIETY HELD AT THE UNIVERSITY FARM, ST. PAUL, MINN, JUNE 25–27, 1935

Through the efficient work of the local committee, headed by E. C. Stakman, a successful series of sessions and field trips was enjoyed by about 60 members and many visitors in connection with the summer meeting of the American Association for the Advancement of Science. No formal scientific papers were presented. An interesting symposium was held on the Past, Present and Future of Plant Pathology, at which H. L. Bolley entertainingly presented high lights from nearly fifty years of experience in this field of science, while E. M. Freeman, looking into the future, presented convincing arguments for the coordination of plant pathological research with related sciences, for the development of more team-work in attacking large or complex problems, and for the maintenance of the autonomous guild or scientific group organization in State or Federal agencies, if the most effective public service is to be rendered in research and regulatory work. Energetic round-table discussions on a wide range of topics at two sessions proved valuable. The Society participated in the joint symposium on Improving the Germ Plasm of Domestic Plants and Animals with Section O of the A. A. A. S., and affiliated societies.

The extensive field, laboratory, and greenhouse investigations at the Minnesota Experiment Station relating to plant diseases and breeding for resistance were inspected, together with an interesting exhibit of current research in the Department of Plant Pathology and Botany. Excursions were made into the region about the Twin Cities to observe plant-disease conditions in the truck, fruit, and ornamental plant-producing areas and at the Fruit Breeding Farm of the Experiment Station. Opportunities for personal conferences were abundant.

By vote of the members present a committee was appointed to work toward the development of a more comprehensive, coordinated program of potato improvement and another committee to work toward coordinated testing of seed-treatment methods. A resolution was passed emphasizing the necessity of adequate, fundamental biological preparation as a basis for satisfactory instruction in plant pathology, and deploring the tendency of administrators in some institutions to reduce the basic biological science requirements. Another resolution was passed to the effect that "The American Phytopathological Society desires to encourage efforts toward laying a sound foundation for an adequate classification of plant viruses. Recognizing that this can be attained only after a thorough study of the

viruses, their properties, their insect vectors, host ranges, their effects on the host plants, etc., the Society desires to encourage cooperation and coordination of effort toward this end.''

At the enjoyable plant pathologists' dinner, a series of skits by the Minnesota members penetratingly interpreted the past, present, and future of plant pathology, and a loving cup was presented to the pioneer plant pathologist, Dr. H. L. Bolley.

HOWARD P. BARSS,
Secretary.

PHYTOPATHOLOGY—AND ITS FUTURE[1]

E. M. FREEMAN[2]

The history of phytopathology's past is well known to this audience. I need not discuss it. The so-called present is merely the recent past. That leaves only the future for me to discuss.

In considering the future of plant pathology in the light and knowledge of the past, it seems to me highly important that we analyze at the outset the important concepts in our collective mind that it includes. To some the future problems revolve about investigational techniques, to others governmental organizations are paramount, and to still others the future personal fortunes of those engaged in this field are most absorbing.

Plant pathology (1) comprises a field of scientific knowledge and investigation; (2) it includes an application of knowledge and skill in the control of plant diseases; (3) in its socio-economic aspects it demands an organization not merely of the scientific facts and practices but of the social, economic, governmental, and professional forces and individuals that contribute to the field and (4) it houses a family of scientists, teachers, technicians, and practitioners that constitute a guild of plant pathologists. Perhaps there are other categories that merit inclusion, but these are outstanding to my mind, and their consideration will consume all of my time in this symposium.

The Science. As a science, plant pathology is inextricably interwoven with the other biological sciences and particularly with botany and its various other subsciences. Perhaps its twin sister may be said to be physiology. More probably botany, that prolific mother of plant sciences, may have given birth to quintuplets whose names were pathology, physiology, mycology, bacteriology, and morphology. They were born in that golden age of the renaissance of botanical science in the middle of the past century. The attending physicians became permanent and brilliant stars in their fields of science; Hofmeister, the interpreter and harmonizer of cryptogamic and phanerogamic morphology; the Tulasnes, discoverers of fungus versatility; Kühn, first to merit the degree of plant doctor; and DeBary, bacteriologist, morphologist, mycologist, pathologist, master mind and botanical savant. Nor must we overlook their colleague, Darwin, who modestly

[1] Read before the opening session of the Summer Meeting of The American Phytopathological Society, held at the University Farm, St. Paul, Minn., June 25–27, 1935.

[2] Chief of the Division of Plant Pathology and Botany, University Farm, St. Paul, Minn.

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expounded the principle around which all of their infant sciences revolved and evolved.

Almost a century of scientific achievement has not altered that close relation of plant pathology with its sister sciences. Rather has the complexity of family relation increased. Ecology, cytology, biochemistry, genetics, biometry, and others have grown to youth or maturity and are contributors to, if not part and parcel of, the field of plant pathology. The enormous expansion and increasing complexity of all of these fields of plant science have steadily constricted the scope of operation of the individual scientist. A DeBary, superman in many fields, is no longer humanly possible, and it needs no divine power of prophecy to predict that he will never again appear. Plant pathology of the future must content itself with a growing intensity of specialization. Paradoxically coupled with this increasing specialization will be found an increasing demand for a knowledge of the literature of the sister and other sciences.

The realignment to these modern complexities has already taken place in the more recent past, and there can be little doubt of the path for the future. Plant pathology must coordinate its research more and more with those sister sciences. The superman must be succeeded by intelligent, generous, genuine, and spontaneous cooperation. Team-work actuated by desire and necessity is the only effective substitute. Because this may be difficult, irritating, or inconvenient is no valid argument against it. Modern complexity in science as well as in social, economic, and political fields demands and must have this increasingly complex organization. The individual who clings to the extreme raw individualism of the scientific past must be content to operate in an increasingly restricted orbit or disappear entirely as an important contributor of knowledge.

I need not elaborate greatly on what that cooperation involves. It means that investigations such as genetics of plant pathogens, biochemical interpretations of disease manifestations, cytological explanations of life-history processes, biometrical studies of pathological phenomena, morphological investigations of host and pathogen are subjects of study for pairs or groups of scientists professionally trained in diverse fields. To the individual plant pathologist it means the careful selection of his particular field of research and study and a cooperating knowledge of those sciences that impinge on his field so that he may intelligently seek and effectively use the aid of specialists in those fields.

One may properly ask: Are we doomed to become workers in a teeming anthill, each with his little load of assigned and tedious labor? I think not. Specialization can best be enriched by a broad knowledge of surrounding territory. The sacrifice of time and effort in going even far afield to become acquainted with the work, the attitudes, the techniques, the successes and

failures in related and even unrelated fields are enlightening and stimulating experiences for every scientist.

The Practice of Plant-disease Control. In the practice of plant-disease control I find myself constantly on guard against unjustified analogies with the practice of medicine among humans and other domesticated animals. These analogies are alluring because of the long history of human medicine and the outstanding achievement of that great group of sciences which underlie it. To my mind the practice of plant disease control is so circumscribed by different conditions that conclusions from any analogies must be scrutinized with the greatest of care.

It is quite obvious at the outset that the practice of human medicine concerns only one biological host species on which centuries of research have builded an incalculable fund of knowledge and in which an interpretation of the responses to treatment are greatly aided by human intelligence. In plant pathology the number of host species is legion. The disparity is further accentuated by the multiplicity of pathogens on each plant host and the modern discoveries in biological forms of pathogens do not at all simplify the matter. Furthermore, the practice of disease control is inextricably bound up with cultural practice, soil conditions, climatic factors, and all of these may vary with each host species. And, finally, many of the host plants may be of such low economic value that the cost of disease control would be prohibitive, while human life is said to be priceless.

The plants that may be said to be of economic significance are not only imposing in the vast number of species, but they are nothing less than bewildering in their diversity. They range from mushrooms to giant trees, from inhabitants of the moist tropical jungle to cacti of the desert; they include producers of foodstuffs in all climates, soils, altitudes, and latitudes; they range from thallophytes to composites.

The great and fundamental difficulty of the application or practice of the several plant science specialties, including plant pathology, in the art of plant production, lies in the basic difference of the vocations involved. The scientist's specialty must necessarily follow some path in the field of his science. He may choose genetics, pathology, or some other special field, but he cannot cover all or many of them.

In sharp contrast to this, the plant grower must confine his efforts to one or a limited number of crop plants. His business never coincides with the activities of one scientific field. He must apply the results obtained by specialists in soils, genetics, plant pathology, and all the other sciences.

Of course, it is conceivable that a new type of trained specialist, such as a plant doctor, eventually may evolve—one who has equipped himself primarily with a knowledge of the pathology of a single crop or a small group of crop plants. Perhaps such a profession may succeed financially as well

as professionally. One shudders to think, however, of the quacks and crooks who will pose as experts in that day. One is reminded of the fake medical cults of today and the difficulties encountered even in controlling them, to say nothing of their extermination. Indeed, we already have a vocation (or is it a profession?) of tree surgery, which has rendered some real service, but in whose name innumerable biological crimes have been committed and to whose coffers has flowed much money extracted from a gullible public.

If we take stock of the status of the application of plant pathology today, we find, perhaps, the closest analogy to human medicine in socialized medicine, the goal of many medical educators. Our agencies of plant-disease control are largely governmental through educational and research institutions. Advice and information in every phase of crop production cost only the effort of asking for them. That service includes the experience of the best specialists in the whole gamut of plant sciences. But the extent and nature of that service are sadly inadequate for the task, even though the state and federal support may be considered generally generous. Perhaps the greatest weakness in our present clinical system lies in the lack of diagnoses in the field and the ignorance of the grower as to his specific needs and the means of supplying them.

To sum up this matter of the future of the practice of plant-disease control, two developments seem probable. Certainly the established clinical methods of experiment stations and extension departments, federal departments, and other government agencies will continue to combine their research activities with the practice of control as in the past. Since commercial specialists in such control have already appeared in our economic order, it seems fair to assume that they will continue. The more probable development of such service seems to be through farm management and similar advisory service corporations large enough to include specially trained men in the several fields basic to the crops concerned. Perhaps the general practitioner or plant doctor may also find a place.

The Organization of Plant Pathology. Research, teaching, and extension of knowledge concerning plant pathology are centered almost exclusively in State or Federal institutions of research and education. Its coordination with other plant and animal sciences in the various governmental and educational institutions is not merely an academic question but one of vital importance. Like every other plant science, it merits prominence and support commensurate with its potential and demonstrated value to agriculture and the general public. Plant pathologists have abundantly demonstrated the value of their investigations and services. Even examples are unnecessary to this audience. Neither is any proof needed of the potential values of this specialized science, which will surely grow with the increasing com-

plexities of our agricultural and economic progress. But the development of any science may be seriously affected by its position in the educational or governmental institution where it functions.

While most large State-supported universities and agricultural colleges maintain separate departments of plant pathology, their subordination, in some institutions, to other coordinate scientific fields seems to me unwise and, for the most part, unjustifiable. Plant pathology has important activities beyond the confines of either horticulture or agronomy. Its subordination to other sciences in smaller institutions or organizations may be unavoidable because of insufficient revenue, but such necessary administrational adjustment should, by no means, restrict the broader activities of the plant pathology staff.

Of greater concern, however, is the administrational development which appears from time to time in State and Federal so-called reorganizations. The organization and coordination of institutional departments and bureaus are not simple matters. There are fundamental difficulties which necessarily give rise to conflicting interests. An analysis of what I have in mind may well begin with the organization in a large university or agricultural college. Since teaching and research are the primary functions of the staff and since extension service is frequently segregated into a separate unit, it follows almost inevitably that the departmental organization follows the lines of scientific specialization. Departments consist of groups of specialists trained in the same professional guild. New departments arise as some subscience specialty attains sufficient importance and prominence to merit an independent and autonomous administrational status.

To my mind the important feature is not so much the catalog status of any given science. The paramount concern should be the best opportunity of guilds of similarly trained scientists with common educational and research objectives to work together to their best advantage. No administrational scheme is worth more than the paper on which it is written if it does not have an understanding and sympathetic administration in charge. No administrational scheme can possibly eliminate the overlapping of sciences and departments. From their very nature they must overlap. It is the chief business of the administrator to see that these overlapping borders are well oiled to prevent excessive friction. Too often the administrator assumes that a reorganization or a dictatorial ukase will eliminate these problems, whereas the chief need is an oil can with a liberal supply of cooperation—spontaneous if possible, compulsory if necessary—and not one drop of isolation in its mixture. While college and experiment station coordinations affecting plant pathology are not without their problems, these seem to me solvable on the basis of our present organizations which, in general, are fundamentally sound.

Of far greater concern, however, are government and State organization affecting the activities and status of plant pathology. Our Federal and State departments of agriculture are basically research or service organizations or both.

The professional guild basis, though historically entrenched here and there, is constantly confronted with the competition of a project system or a crop system of organization. I am not one to claim a sufficient wisdom to criticize such organizations as universally inappropriate or inefficient. But all these bureaus and departments are fundamentally grounded in the sciences which solve their problems. Whatever may be the administrational device used to accomplish the necessary ends, two things stand out in my mind as fundamental. First the recognized professional guilds must be maintained in sufficiently large groups to furnish conditions under which they may cooperate and maintain their guild existence. Plant pathology is one of these recognized groups. An organization that subordinates whole professional guilds to a single crop plant or commodity project may thereby demote professional scientists to skilled labor functions and may so delimit the scope, activity, and service of such scientists that inexcusable duplication is encouraged and time and money-saving cooperation discouraged. It is basic to the nature of governmental service in agriculture that the horizontal demand for service in all fields of science is crossed at right angles by the scientific guilds that render that service. No organization devised by man can possibly change that fact. The solution lies not in cutting either the horizontal or the vertical lines. It lies in the administrator's skill and responsibility to see that both lines are kept intact with the least possible confusion and friction. It is his business, for which there can be no substitute scheme of organization, to coordinate in a horizontal plane the activities of all of the scientific guilds to the use of the growers of one crop plant. It is equally his business in the interest of public welfare and economy to maintain the efficiency, autonomy, and cooperative facilities of each of the important scientific guilds that give their service to that crop plant or to related crop plants. Even in a broader field it seems to me this principle ought to apply. Repeated attempts have been made to remove the United States Forest Service from the Department of Agriculture to the Department of the Interior. Such removal would be a wholesale separation of the numerous scientific guilds that serve forestry from their close contacts with the closely allied guilds of the field of agriculture of which they are an integral part. The recent removal of the Soil Erosion office to the Department of Agriculture seems to be at least a second-thought recognition of what is obviously the sound coordination of that work.

Of all the problems that confront plant pathology, one of the most difficult and perplexing is that of the quarantine and regulatory control

of plant diseases and insect pests. To my mind this problem offers fundamental difficulties that are not always appreciated. While I am firmly convinced that the principles of organization stated above should apply wherever possible, there are other factors that cannot be ignored. Chief among these factors is the legal nature of the enterprise. Quarantine and regulation are neither research nor education, though both of the latter may be valuable aids. They are laws and require enforcement. A new guild enters the picture. Indeed, the enforcement of the law is the primary objective and would seem to demand a preferred position in organization for administration. It is obvious that in the Federal control, where the Department of Agriculture has been faced with regulatory activities in almost every field of agriculture, that the administration of large groups of regulatory activities be concentrated. After all, regulatory work may be a distinct detriment when coupled too closely with publicly supported research and educational institutions.

But there is another side of this problem that is of particular interest to the future of plant pathology. Plant quarantines and regulatory acts frequently involve plant diseases as well as insect pests. Regulatory administration must have the aid and assistance, if not the actual direction, of trained scientists. The importance of plant diseases in the regulatory field is sufficiently large to justify a greater participation from the guild of plant pathologists. Regulatory administrations, I believe, increase their efficiency by observing the principles of guild autonomy discussed above. It is to that end that pathologists should aim, both in the interests of their own science and for the improvement of public service.

The Guild of Plant Pathologists. Plant pathology has merited and won recognition as a plant science throughout the world. Its importance in the future cannot diminish but must increase. With the inevitable and increasing complexity of agricultural industries will come more and greater specialization within the guild. But that specialization involves not so much a narrowing of training within the science itself as an intelligent coordination with sister plant sciences. New fields of professional activity have already opened up. Many more will follow. Their number and importance will depend largely on the initiative of the future members of the guild. Success will depend on their understanding not merely of the scientific facts involved, but also on a grasp of economic conditions and currents. It will require the application of scientific training to public and private enterprises. It will need scientific imagination coupled with practical knowledge and common sense. It will offer a career in fundamental research, pure and undefiled or gloriously practical. It will offer a satisfying field of combat against one of the most potent, destructive forces of nature.